

TRANSACTIONS
OF THE
Association of
Life Insurance Medical Directors
of America

FIFTY-FIFTH ANNUAL MEETING

Harry E. Ungerleider, M. D.
Editor

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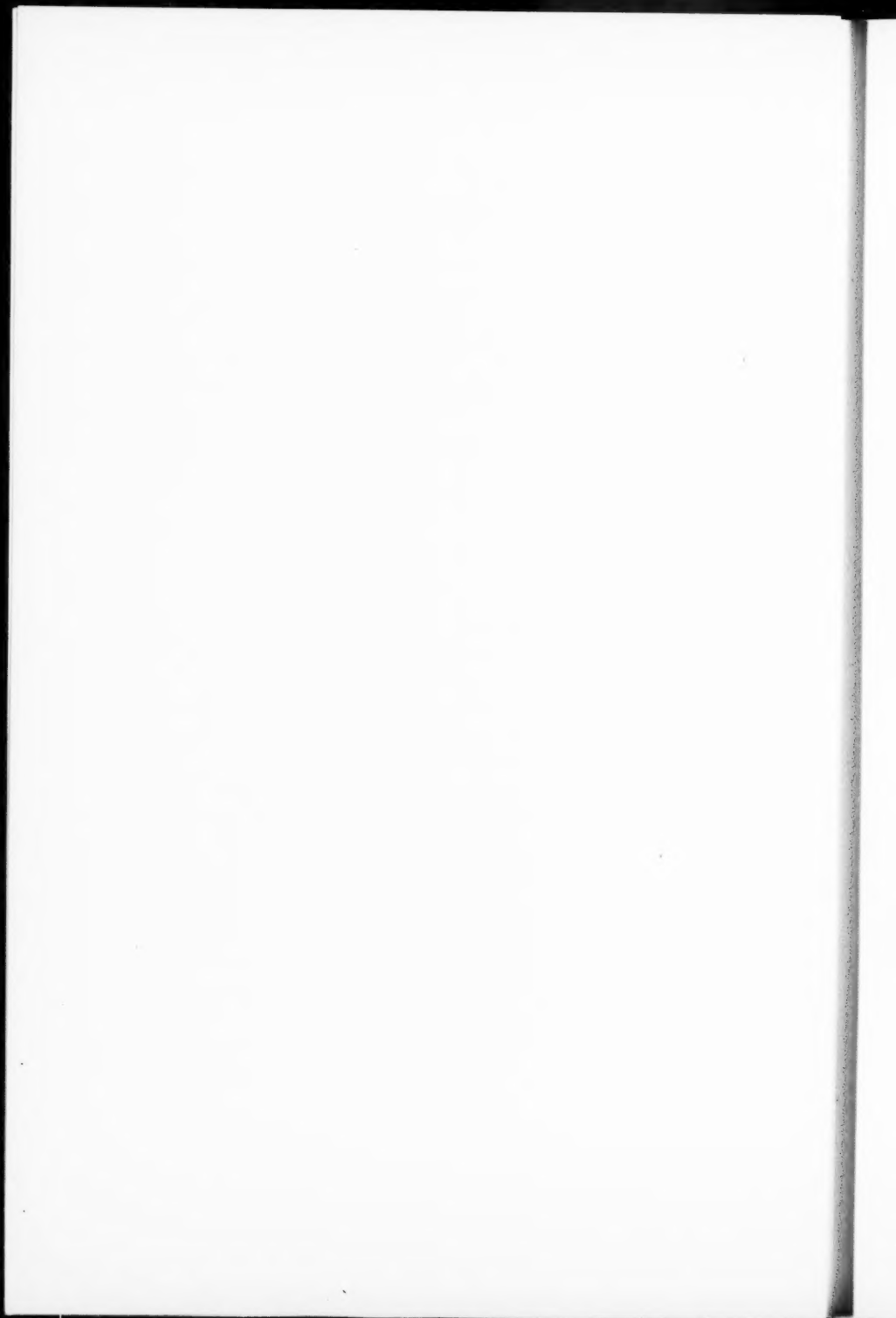
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DEDICATION

Although this volume is designated the thirtieth in the series of the Transactions of the Life Insurance Medical Directors, there has been a lapse of four years since Volume XXIX was issued. Several attempts were made to hold scientific meetings of the Association but due to the war and governmental restrictions, this could not be done.

Dr. William Bolt, Chief Medical Director of the New York Life Insurance Company, was President during this period. Although no scientific meetings were held, matters of a scientific and business nature were considered both by the council and business meetings of the Association. During this period, the Constitution and By-laws were revised. The initial meetings in conjunction with the Life Insurance Medical Research Fund represented one of the outstanding activities and Dr. Bolt played a leading part in these discussions.

This volume is dedicated to Dr. Bolt as a tribute to his leadership in forging the strong links between the prewar and postwar periods in Life Insurance Medicine.



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FIFTY-FIFTH ANNUAL MEETING

The Fifty-fifth Annual Meeting of the Association of Life Insurance Medical Directors of America was held at the Hotel Pennsylvania in New York City on Thursday and Friday, October 24 and 25, 1946.

PRESIDENT STREIGHT—It is my very pleasant duty to call this fifty-fifth annual meeting to order. For selecting me as your President I ask you to accept my warm thanks. To serve you and this Association has been an honor and a pleasure as well. Your Executive Council and various committees have worked so well that my duties have not been arduous.

To our many new members, guests and delegates we offer a warm welcome. I hope that during this meeting each of us may form new and enduring friendships. Let us all try to make this meeting as memorable for the warmth of its fellowship as for its interest in the papers presented.

The programme arranged, I am satisfied, will well reward our earnest attention. It is practical and timely and it is hoped that from it we may all gain fresh inspiration and new ideas which may well broaden the horizon of our decisions. We hope that many of you will feel impelled to join in discussion of the papers presented. It is important to remember that the kernel of interest may well be further developed and clarified by the free exchange of ideas and opinions from the floor. If you have a question to ask or additional information to present, let us hear from you.

Our last scientific meeting was held under the cloud of war and at a time when the fortune of the allied nations was at its lowest ebb. While it was, I am sure, the firm belief of each of us that eventually our enemies would be "beaten with many stripes and cast into outer darkness", it was beyond human vision to foretell when that day would arrive. It eventually arrived with such suddenness that we could scarcely believe it. This year, 1946, finds the struggle for the freedom of the world ended, only to find that the problems of peace are not less difficult to resolve than those of war or than those of peace have been at any time throughout man's long and continuous endeavor to the end that there should be more happiness and comfort in life for everyone.

The purpose of this Association is the promotion of medical science as applied to the selection of risks, to understand and interpret the significance of disease in its relation to the continuance of life. This requires the widest range of medical knowledge and experience. It is important that our decisions should be in accord with recognized clinical opinion and that they should appear reasonable to our examiners who frequently are called upon to interpret our action to both applicant and agent. We must endeavor to judge fairly the evidence before us. Judgment is the faculty which enables one to arrive at a wise conclusion, sometimes upon imperfect evidence. It is the one talent which no rules will teach, and which knowledge and experience do not always give. It is the capacity to see things as they are and of making a wise choice between different points of view. Through the exercise of sound judgment in our decisions we shall inspire confidence and create good will.

While the institution of life insurance had its birth in the British Isles, it has seen its greatest growth and development in the United States of America and Canada within the present century. Today it has reached such proportions that every second person in our two countries is insured. Where there is such widespread interest, there is an associated responsi-

bility for service beyond the conditions in the contracts. It is, therefore, a real pleasure to report that during the past year the life insurance companies have established The Life Insurance Medical Research Fund, of which Dr. Francis R. Dieuaide, the Scientific Director, will speak later. In this combined research programme the life insurance companies have taken a progressive forward step, the results of which can scarcely be imagined and may well exceed our greatest expectations. By combining our efforts in this endeavor to search for the cause and development of disease, we have an opportunity to contribute in a significant way to the total of human happiness and health. Of the significance of health, need we say more than that it paves the way for increased happiness and performance, and makes for more effective individual and national life and labor, and so helps production and prosperity. It provides, in fact, our best national economy.

The institution of life insurance enables more people to serve themselves and to better purpose, than is possible by any other existing means. It is the greatest institution for thrift which man has devised. It is deeply interested in all that pertains to human happiness, comfort and general well being. It is the outstanding example of the benefits which may be secured through co-operation and good will. It is built upon faith, confidence, fidelity and security, and it is more and more becoming synonymous with fidelity and security. To set it apart from other forms of endeavor, it has the well-being and happiness of mankind as its major incentive. Welfare connotes security, recreation, friendly association, something to think about, something to take pride in, and the need to matter to someone. It implies human concern, being cared for, and this is what most people often need to give meaning and fulness to their lives. Men will work more freely, more contentedly and more productively when they have established so far as possible security for themselves and their dependents. Life insurance, the outstanding example of co-operative effort, is the avenue through which this may be achieved.

During the war years many new ideas for the diagnosis and treatment of disease were advanced, perhaps the most outstanding being the discovery and development of penicillin by Fleming and Florey. Many other outstanding advances were made in the treatment of wounds and disease with great saving of life and lessening of disablement. In spite of this progress it is disappointing to observe that casualties on the home front during the same period exceeded by many thousands the loss of life and disability caused by war. This is a disturbing fact which calls for serious thought and concerted action by everyone interested in human welfare. Many untimely deaths occur each year from preventable causes. Preventive medicine and accident prevention are important fields to which life insurance companies may well give more attention with definite benefit to their policyholders, and in a broader application with distinct advantage to national economy and human welfare.

The outstanding position which the institution of life insurance holds today reflects in a considerable degree the application by those responsible for medical selection of the principles embodied in a broad knowledge of human nature and its probable reaction to a changing environment. That which life has taught us, our knowledge, is a loan only, which we must pass on to others, having enriched it by the result of our study and experience. To those of you who have more recently entered the field of life insurance medicine, may I suggest a careful study of the published Proceedings of this Association. These volumes contain a wealth of clinical and statistical information which should be of immeasurable assistance to you in adjusting your clinical ideas to the evaluation of risks.

There are many conditions which effect longevity and which await more exact observation and description. The process of ageing is an example. What is the upper limit of normal age? What is normal ageing? Have we any dependable or positive evidence of the factors which cause premature ageing?

If so, what are we doing or what can we do about this? Should our concern be to the end that life should be more completely satisfying, — rather than merely added years — that is to add life to years, not just years to life.

The future with its promise and opportunity lies ahead. We must constantly strive to continue and enlarge the ideas of those who organized this Association and whose memory we may best honor by well directed efforts to extend the benefits of life insurance to a gradually increasing number.

On behalf of this Association I wish to extend to those who are here as delegates from the Medical Section of the American Life Convention a very warm welcome. We are glad to have you with us. We invite you to enter freely into the discussions. We wish to know you better and that you should know us better.

I believe Dr. Raymond B. Hutchinson, Chairman of the Medical Section of the American Life Convention, has a message for us. Dr. Hutchinson!

DR. HUTCHINSON — It is a pleasure to bring you greetings from the Medical Section of the American Life Convention and to extend to you a cordial invitation to attend our next meeting.

This will be a three day meeting with our mornings given over to scientific sessions and in the afternoons we will have the opportunity of golfing, swimming and many other sports that this marvelous spot at Asheville offers, not to mention the opportunity of renewing pleasant associations with our friends. The meeting is to be held at the Grove Park Inn, Asheville, North Carolina on May 28, 29 and 30. I hope every one of you will make an effort to come down and enjoy the Southern hospitality with us.

PRESIDENT STREIGHT — Dr. Francis R. Dieuaide, Scientific Director of the recently organized Life Insurance Medical Research Fund, will now tell us of the progress made in the first year of the Fund's existence. Dr. Dieuaide!

DR. DIEUAIDE—Mr. President, Members of the Association of Life Insurance Medical Directors: I am deeply grateful for the privilege of addressing you on this important subject and I shall be glad to answer any questions you may wish to ask regarding it.

LIFE INSURANCE MEDICAL RESEARCH FUND

BY FRANCIS R. DIEUAIDE, M. D., *Scientific Director*

The members of this Association all know that the Life Insurance Medical Research Fund was first organized just one year ago, with 143 companies as charter members. There are now 148 company members. The constitution of the Fund vests control of its operation in a Board of Directors who are assisted in matters of scientific policy and in the selection of projects for support by an Advisory Council composed of eminent medical scientists, all of whom have long experience in planning and evaluating research.

During the past year, a Scientific Director has been appointed, an office has been established at the Academy of Medicine in New York City, methods of procedure have been worked out, and a number of grants have been made.

You are probably aware that the Fund is devoted for the present to the support of fundamental research on problems of cardiovascular disease and function. Some such definition of scope is necessary in order to make possible a coherent program and to prevent inefficiency. The cardiovascular field is one of obvious importance to life insurance companies and their policyholders, as well as to the public. Its choice as the working ground of the Fund is widely regarded by medical investigators as a wise one. With the generous help and sound advice provided by the Advisory Council, the Fund is being wisely put to work.

Two methods of aiding research have been adopted. The Fund itself is not attempting to conduct investigations. Instead, grants of money are being made to institutions which have the necessary key scientists and the basic facilities. The second method of working is through fellowships which are granted to promising young individuals to enable them to obtain training and experience in research. The need for increasing the number of qualified workers in this field is particularly great in this postwar period and will probably continue to be so.

Only a small percentage of the funds will be used for administrative expenses.

The sum made available to the Fund in the last quarter of 1945 and the full year 1946 is close to \$735,000. A total of 54 grants with a value of \$633,591 have already been made. These grants were made to 31 different institutions in 20 states and Canada for the support of specific research programs. The projects supported cover a wide range from studies of normal function in areas in which present knowledge is seriously defective to investigations on the prevention and treatment of cardiovascular disease. Since none of these grants has yet been in effect for a complete year, it is impossible to give any account of the results at this time.

Fellowships, with a total value of \$55,800, have been awarded to 20 individuals from various parts of the United States and Canada. These 20 Fellows are now engaged in cardiovascular research under the supervision of experienced investigators at as many different institutions in both countries.

The Life Insurance Medical Research Fund has attracted wide and favorable attention from the Medical Profession and the public. I believe we are justified in saying that it has made a good start. Before advances of practical value in the prevention and treatment of disease can be made, much spadework has to be done on the causes and mechanisms of disease and even on normal function in order to fill up wide gaps in our knowledge. For this reason, medical research progresses only at a slow rate and it is too much to hope for practical results at an early date. We may be sure, however, that the Fund will be fruitful. In the meantime, it seeks your full support, which I believe it deserves to have.

PRESIDENT STREIGHT—We now come to the regular program and Dr. T. Duckett Jones, Director of Research in Rheumatic Fever and Rheumatic Heart Disease, House of the Good Samaritan Hospital, Boston, Massachusetts, will address us on a subject on which he is so well qualified to speak "Rheumatic Fever". Dr. Jones!

RHEUMATIC FEVER

BY T. DUCKETT JONES, M. D.

House of the Good Samaritan, Boston, Massachusetts

One may assume that your organization has a real interest in rheumatic fever, and even more interest in the resulting rheumatic heart disease. This is inevitable since it figures so consistently in the evaluation of risks and the elimination of poor risks who apply for insurance. I believe that some of the features which influence prognosis and information gathered through long observation of such patients may be worth your consideration. Hence, I wish to summarize observations on the natural history of rheumatic fever with additional remarks concerning the application of our present knowledge for the sufferers from this disease.

Reports concerning rheumatic fever and heart disease are numerous. In the past thirty or forty years there has been much interest in this problem, particularly on the part of internists and cardiologists. One of the most interesting reports concerning prognosis is that of Grant¹, the study comprising the 10 year observation of 1,000 men with heart disease. Rheumatic heart disease occupied an important part of this group. Grant's study is selective in certain ways: All patients were males; all were veterans of World War I; and there was little information concerning the early health of this group. In other words, the disease pattern had to some extent defined itself before the period of actual observation.

Clinical observations on the patients in this report have been made by physicians and house staff of the House of the Good Samaritan. Dr. Edward F. Bland and the author have taken part in the observations and conducted the analyses. Doctors Benedict F. Massell, George P. Sturgis, Howard B. Sprague, William David Smith and John P. Hubbard have consistently aided in the clinical observations.

Other important studies have been highly selective also. The recent excellent report of Cohn and Lingg² is composed of data on rheumatic heart disease individuals all of whom came to autopsy. The material is large and the information important, but the study is perhaps the most selective of all. Conclusions are of necessity tempered by the criteria used and the selection of material. If only fatalities are to be evaluated, one may obtain a false value of prognostic features. The value of the application of the best statistical methods is dependent upon the choice of material selected and the pertinence of this selection to the desired knowledge. One of the serious difficulties in evaluating rheumatic fever and rheumatic heart disease literature is the fact that most reports have included average periods of follow-up, rather than a similar period for each patient included. Four or five such reports lose much of their effectiveness in the method of analysis and, hence, in the interpretation of results.

The actual material which I wish to present to you is again a selective series. It would be best to define the selection at once. The material represents 1,000 rheumatic fever and chorea patients given care at the House of the Good Samaritan. The initial data on this group have been reported briefly by Bland³ and myself. The 1,000 patients had been followed in each instance for 10 years, at the time of the initial report. The rheumatic fever diagnostic criteria have been reviewed⁴ elsewhere. The criteria for the diagnosis of rheumatic heart disease were essentially as described in the Nomenclature and Criteria for Diagnosis of Diseases of the Heart by the New York Heart Association, New York, 1940. All of the patients at the time of initial rheumatic fever or chorea were under 21 years of age. This purposelessly excludes the group of rheumatic fever or heart disease patients who first come to medical attention in adult life. This may represent the exclusion of one-third of all of the rheumatic fever and heart disease patients. There is but scant knowledge concerning the volume of rheumatic fever developing initially in adult

life. The average age at onset of the group was 8 years. There was a heavy loading on the side of females, not because of any greater incidence of rheumatic fever in the young female, but because of the selectivity of our hospital service. There was no selectivity on the basis of the severity of initial rheumatic fever or chorea. Many studies indicate that there is no significant difference in incidence of the disease in the two sexes in early life. The younger rheumatic fever and heart disease patients have been chosen for definite reasons. It has long been known that the majority of the disease begins between the ages of 5 and 15 years. We wished to observe the disease from its beginning and follow the patients for as long a period as possible. This would give us our best opportunity to observe the natural history of the disease.

Previously published data on this group presented information of some interest. It may be well to review the chief points. Using very strict diagnostic criteria both for the diagnosis of rheumatic fever and for the presence of rheumatic heart disease, it was found that two-thirds of the patients had some evidence of rheumatic heart disease at the time of the initial attack of rheumatic fever. Approximately one-third of the group was free from any detectable heart disease during the first attack. We designate this group as having potential rheumatic heart disease (P. R. H. D.). At the end of ten years the outcome in these two groups was dramatically different.

Of those without evident heart disease (342) at the time of the initial attack, 70% remained free of evident heart disease ten years later. Only eleven individuals in this category died within ten years, and in four of these, death was unrelated to rheumatic fever or heart disease. Seven of those dying developed severe rheumatic heart disease as the result of recurrent rheumatic fever. Approximately 25% of this group developed some evidence of rheumatic heart disease in the course of ten years. Hence, P. R. H. D. at the onset affords an excellent outlook, and the absence of heart disease in the first attack

of rheumatic fever or chorea may be considered favorable for the patient's prognosis.

Those individuals with rheumatic heart disease (658) at the initial attack did less well: 29% of this group died within ten years; 27% had no change in their rheumatic heart disease status; 21% had a progression in the evident severity of heart disease; and 22% had a regression or disappearance of evident heart disease. Distinct improvement in the latter group (22%) is of considerable interest. They represent approximately 15% of the total group of 1,000 patients. In one-half of these there was a complete disappearance of demonstrable rheumatic heart disease, the remaining half showing a decrease in evident rheumatic heart disease as indicated by heart size, disappearance of significant murmurs, or improvement in heart function.

As indicated above, death from rheumatic fever and heart disease in the first ten years occurred frequently in the group having rheumatic heart disease with the initial attack. While approximately 20% (203) of the total group died within ten years, there were 19 (9% of dead), who died of causes unrelated to rheumatic fever or heart disease. In 16 (8% of dead), death was due to bacterial endocarditis. In the remaining 168 (83% of dead), death was due to recurrent rheumatic fever with heart failure as a terminal development. Strangely enough there were a number of accidental deaths in this group and several patients died of tuberculosis.

The ability for ten year survivors to carry on normal physical activity was surprising. Two-thirds of the total group (648) were able to lead physically active lives. Approximately one-third of the total group (313) had no evident heart disease. An additional one-third of the total group (335) had some degree of detectable heart disease but not sufficient to interfere with good cardiac function. Some of the individuals in this group were advised to avoid competitive physical activity because of anxiety and without any certain proof that such activity would be deleterious to them. Of particular interest

is the fact that 313 (31.3%) of the entire group had no evident heart disease ten years after the onset of their disease. This problem of physical activity and prognosis is of considerable importance and will be mentioned later on.

It is obvious that in addition to the large number dead (203), a reasonably large group must be expected to develop sufficient heart disease to interfere with physical activity. This group numbered 135. Physical activity in this group varied considerably but in a fair number of instances cardiac reserve was so diminished as to require almost a bed-chair existence. In some patients active rheumatic fever and progressive rheumatic heart disease persisted for some years. Others were able to carry on reasonably well provided they avoided strenuous exertion. This group has distinctly diminished cardiac reserve. Considerable cardiac enlargement was consistently present. These patients are of interest to cardiologists and frequent the medical wards of general hospitals. They rarely live beyond middle age. Auricular fibrillation is a frequent development, and varying degrees of chronic cardiac failure are usual. We can expect to benefit these patients but little. They represent cardiac cripples and a serious economic loss.

To briefly review the first decade results of the 1,000 patients, one may say that the outlook was found to be poor in a total of 338 (33.8%), since death occurred or obvious cardiac crippling developed in this number. The remaining two-thirds (648), were able to lead active lives and their ultimate prognosis seemed dependent upon the occurrence of subsequent recurrent rheumatic fever or bacterial endocarditis.

The survivorship rates of this group of patients has been worked out for us by Mr. Herbert H. Marks of the Metropolitan Life Insurance Company. Mr. Marks' data shows that in those individuals without evident heart disease at onset, 96.7% survived the ten year period. There was no difference in the sexes in this group. Of those with heart disease at the initial attack, 72.1% survived the ten year period with

little, if any real difference between the sexes. There was little if any significant difference in the survival percentages on the basis of the age of the onset of the disease by five year periods up to 21 years of age.

During the ten year period, 70% of these patients had more than one attack of rheumatic fever or chorea.

Valvular lesions were of considerable interest. As expected, mitral disease of some degree was the common finding. There was, however, a significant predominance of the severer degrees of mitral disease in the female. This was especially true of so-called 'pure' mitral stenosis. Evidence of aortic valve disease occurred in approximately one-half of all patients with rheumatic heart disease. At the end of ten years a significant number of females were found to have evidence of slight degrees of aortic regurgitation. However, there was a very striking preponderance in the male of so-called 'free' aortic regurgitation (with peripheral evidence of increased pulse pressure).

Other striking observations may be summarized: 1. There was little correlation between heart size and evident valvular disease. 2. Prognosis was to a considerable extent dependent in the first decade on the unpredictable severity of recurrent rheumatic fever. 3. Recurrences of rheumatic fever or the absence of such recurrences obviously influenced the ten year results, both as to mortality and the degree of permanent heart disease. This would seem to indicate that it is difficult to make a reliable prognosis prior to adolescence. 4. Heart size, as in the study of Grant¹, proved to be of considerable prognostic importance. Those individuals with considerable degrees of cardiac enlargement did poorly.

This group of 1,000 patients has been followed well into the second decade⁵ of their disease. Approximately 25% of the group have been followed for more than twenty years, in some instances for more than twenty-five years; 50% have been followed for between fifteen years and twenty years; the remaining 25% have less than fifteen years of follow-up.

This latter group can be easily brought to the 15 to 20 year observation period by concentration on their records of the last several years. A fair number of these patients were in military service and their data have been difficult to obtain. As yet second decade data have not been well studied as the decade is yet incomplete, observations being now in progress. The most active observer of the group (Bland) has been in military service. Further, it has been difficult to obtain funds for the varied clerical and other services which are essential in the accumulation and analysis of data on a large group of individuals. Unfortunately the natural history of rheumatic fever and heart disease has not been of sufficient interest for any of the large foundations to continue active support of observations over many years.

However, some additional information concerning this group may be of interest to you, since the average period of follow-up is approximately 18 years at the present time. Further evidence of the severity of rheumatic fever and rheumatic heart disease is indicated by the fact that an additional one hundred patients have died since the completion of the ten year study. This raises the mortality in this long study period to 30%. Apparently 660 of the 1,000 are known to be living at the present time. We have been unable to obtain reliable data on 36 of the total 1,000. It seems likely that most of these 36 are living, as it is easier to obtain data on those who no longer survive. Approximately 75% of those living are still capable of leading a physically active life. In approximately 25% of the living there is a marked reduction of cardiac reserve and inability to do much physical activity. The problems being observed in this group of patients at the present time are very different from those of the first ten years. Pregnancy has assumed a reasonably prominent role. Many of those with little or no heart disease are carrying on work requiring extensive physical exertion. It has been rather striking over the years that physical activity in patients without decrease in cardiac reserve, has produced little if any evident deleterious effect on the patient. This must be quali-

fied to omit the question of appreciable physical activity in the presence of active rheumatic fever.

Of those individuals with potential rheumatic heart disease at the onset, 87% are known to be living. This further strengthens the good prognosis, if no heart disease was observed at the initial attack. 7% of this group are known to be dead and of the survivors, only 40% have any evidence of rheumatic heart disease, the remaining 60% still being noted as potential rheumatic heart disease.

Of patients with rheumatic heart disease at the initial attack, only 54% are known to be living and 43% are known to be dead. Of the survivors in this group, 75% have some evidence of rheumatic heart disease. The proportion of the survivors in this group with obvious reduction in their cardiac reserve is much greater than those who had no evident heart disease at the onset.

Bacterial endocarditis has not developed as frequently in the second decade as we had expected. The number has grown from sixteen in the first ten years to thirty-one at present. It is of interest that in this nearly twenty years of observation, bacterial endocarditis occurred in no single instance in an individual without evident rheumatic heart disease prior to the bacterial valve infection. In the past several years, the use of penicillin has altered the outlook of this previously fatal infection of the heart valves. Since the institution of this type of treatment, no patient in this series has died of bacterial endocarditis.

Mitral stenosis has been an interesting feature of the study. One readily accepts the fact that mitral stenosis is common in those rheumatic heart disease patients who frequent the medical wards of our general hospitals with markedly diminished cardiac reserve and cardiac failure, so often fatal during mid-life. A considerable degree of cardiac enlargement is to be found in these individuals. Mitral stenosis and a considerable degree of cardiac enlargement may occur in either sex.

Of more interest is the fact that we see frequently in the female, mitral stenosis with little or no evidence of cardiac enlargement. Such patients often do well unless recurrent rheumatic fever develops, in which event the heart may enlarge considerably and the patient develop failure. In the female an occasional patient with obvious mechanical difficulty from mitral stenosis is seen, in the absence of appreciable cardiac enlargement. This group has acute pulmonary embarrassment as a rule resulting from emotional experiences or sudden demands on the heart. It is rarely, if ever encountered in the male. This striking difference between the male and female in relation to so-called 'pure' mitral stenosis is of importance. It offers a lead for further studies on factors influencing the natural history of rheumatic fever.

Chorea has been an interesting feature of this series. Approximately one-half of all patients had chorea at some time. In the second decade, chorea was strikingly absent. It did not occur after adolescence in patients of either sex. It is our distinct impression that those individuals who had chorea did better than those who had other and more definite manifestations of rheumatic fever. There was a total of some 116 patients who throughout the period of observation remained free of other manifestations of rheumatic fever than chorea. For convenience, we have designated these patients as having 'pure' chorea. Of these 116, only 19 have developed any evidence of heart disease. This group is predominately female. It has been rare indeed for a male with 'pure' chorea to develop heart disease. Thus, males with 'pure' chorea can be given a good prognosis. Usually they are able to lead a normal life without detectable heart disease years later. It seems evident that there is some striking difference between chorea patients and those rheumatic fever patients without this symptom complex. Further, there seems to be a difference between the male and female chorea patient. This is of some prognostic importance.

Inferences are frequently made that early diagnosis and

proper care will distinctly influence the outcome in these patients. I am sure that you would like to have this matter discussed, or at least an opinion expressed concerning our ability to prevent rheumatic heart disease. I regret to say that I cannot answer this question. I know of no series or study that has yet proved that early diagnosis and proper care have markedly influenced the appearance of rheumatic heart disease or of the ultimate amount of heart disease which an individual may develop. I do not infer that early diagnosis and good care do not help, but there are many aspects which have eluded evaluation up to the present. Exposure to streptococcal infection is one such factor. Our knowledge is far from complete in relation to the evaluation of attempts to protect these patients from respiratory infection. However, you will agree that these patients should have the benefit of the application of our present medical knowledge. We need not only to apply this knowledge, but learn to evaluate the results of our efforts to alter the disease.

Doubtless you are interested in heart murmurs and their interpretation and significance. Many diagnostic mistakes continue to be based on murmurs alone. These could be obviated with ease in most instances. Save in unusual cases, murmurs are less important than heart size. Despite this, murmurs continue to play a leading role in our teaching and evaluation of heart findings. In view of our extensive 'murmur' experience, some murmurs commonly found in rheumatic heart disease patients or troublesome in the evaluation of the presence or absence of heart disease may be discussed:

1. *Systolic Murmurs*

(a) *Significant Apical Systolic Murmurs.* We believe such murmurs to be quite characteristic. They often appear within a few hours of the onset of rheumatic fever. They rarely vary during the course of active rheumatic fever save in the early stages. It has been our experience that systolic murmurs concerning which there is appreciable difference of

opinion among the members of our staff, rarely prove to be of importance. These murmurs are usually at least grade II or III by the Levine Classification. They begin early in systole and are descendent, usually lasting throughout systole. They vary little with position. They are widely transmitted, are not appreciably influenced by respiration, and usually are heard at the lung bases. Such a murmur in the presence of symptoms suggestive of rheumatic fever is strong corroborative evidence that the individual has some heart involvement and that rheumatic fever is the correct diagnosis. Such murmurs have to be carefully interpreted along with other features. Some degree of cardiac enlargement is usually found during active rheumatic fever if such a murmur is heard. Frequently the first sound of the heart will be masked, although sound recordings show that the first sound is not actually displaced. This type of murmur may be caused by mechanisms other than rheumatic fever, such as profound anemia. While we rather freely make a diagnosis of mitral regurgitation or incompetence in the presence of such a murmur, it is not actual proof of valvulitis. It is not indicative or proof of acute or active endocarditis. However, we do know that mitral valvulitis is usual in rheumatic fever and rheumatic heart disease patients. Such a murmur may result from so-called ring changes, involving the base of the valve. In a patient with present or past rheumatic fever, we believe such a murmur is indicative of rheumatic heart disease. Once this murmur appears it usually will be present for a long time, even in the patient who does well and has an ultimate disappearance of this and other evidence of rheumatic heart disease.

(b) So-called Functional or Physiological Murmurs. These murmurs are often confused with those of more definite heart disease significance. They are more often basal in maximal intensity and may be very loud, and at times even harsh. Such murmurs vary considerably with position, and often may be heard in one position and not in another. They are usually influenced by respiration and are rarely heard at the lung

bases. Such murmurs in the presence of a fast heart rate are common in children. They are commonly heard first during respiratory infections. Low-grade fever and such a murmur are often the bases for an incorrect diagnosis of rheumatic fever and heart disease. This is a frequent abuse of diagnostic criteria and the cause of much unnecessary anxiety. In addition to this type of murmur unimportant systolic murmurs are often heard maximally at the apex. Such murmurs usually begin late in systole, are rarely widely transmitted, may vary with position and are not often heard at the lung bases. It is our belief that such murmurs should be disregarded as evidence of heart disease.

The mechanism of these functional or physiological murmurs is uncertain. There are doubtless various causes. They occur in many children without other evidence of heart disease and without rheumatic fever. Many observers believe they are extra-cardiac in origin. Such murmurs are not infrequent in patients with rheumatic fever and abundant evidence of rheumatic heart disease. Even here they seem to be of no importance.

2. *Diastolic Murmurs*

(a) *Mitral Diastolic Murmurs.* In general these are of more significance than are systolic murmurs. The initial mitral diastolic murmur in rheumatic fever is a rumble occurring in mid-diastole, frequently following a third heart sound. In the past this murmur has been interpreted as being indicative of mitral stenosis. We now know that this is incorrect and that it may not be indicative of any real mitral disease. Some observers believe the murmur is the result of a third heart sound plus an auricular component. A large percentage of such mid-diastolic rumbles disappear as the patient recovers from rheumatic fever. Neither is the presence of such a murmur indicative of the likelihood of the ultimate development of mitral stenosis. I have never seen such a murmur in a rheumatic fever or heart disease patient who did not also have a

significant apical systolic murmur. This most frequent mitral diastolic murmur occurs only in individuals in whom there is other evidence of rheumatic heart disease.

The characteristic and important mitral diastolic murmur of rheumatic heart disease is the murmur of mitral stenosis. We never make such a diagnosis unless there is a pre-systolic crescendo phase, save in the patient with auricular fibrillation or congestive failure. Observers disagree as to the necessity and importance of the presence of a systolic click or snapping first sound. Observers disagree also as to the significance of an accentuated pulmonic second sound. For convenience, we have designated as 'pure' mitral stenosis those cases in which such a murmur occurs in the absence of any systolic murmur. Such a finding is not uncommon. This murmur may be long and extend throughout diastole. In other instances, it is of short duration and heard only late in diastole. In rare instances, we have seen such a murmur develop during rheumatic fever of about a year's duration. As a rule, at least two or three years elapse between the onset of rheumatic fever and the appearance of such a significant diastolic murmur. Often the interval is five or ten years. This is the characteristic murmur of insidiously developed rheumatic heart disease, and without previously recognized rheumatic fever. We doubt that auscultatory findings are a reliable index of the extent of actual mitral valve obstruction. As previously indicated this may be determined by function, particularly by the competency of the pulmonary system under stress and perhaps by auricular size by fluoroscopy.

(b) Aortic. Fully one-half of all rheumatic heart disease patients develop murmurs indicative of some aortic valve involvement. The characteristic murmur is, of course, an early, blowing diastolic murmur heard best upon forced inspiration and along the left sternal border. It may be of any degree of loudness and has qualities quite similar to those of the significant apical systolic murmur. It is usually descendent in quality and will frequently last throughout diastole. When aortic

regurgitation or incompetency is considerable, the aortic second sound may be masked or displaced. So-called 'free' aortic regurgitation, with peripheral evidence of a high pulse pressure, is not uncommon in this series. It may develop quickly. Rarely 'free' aortic regurgitation develops in a few weeks, and even suddenly in the event of such an unusual occurrence as an inverted valve cusp. However, usually this is a slow process. In rheumatic heart disease there is no correlation between 'free' aortic regurgitation and hearts of very large size. In some of our patients with 'free' aortic regurgitation, there is little cardiac enlargement. This is contrary to the usual experience with aortic regurgitation from leutic aortic disease. In such instances, the heart is usually very large. It has been rather surprising that while aortic disease seems to be more common in the male, we have a reasonably large number of females with slight degrees of aortic regurgitation.

Aortic stenosis has not been common in these patients up to the present time. Perhaps it will be found more often as the patients age. It has occurred more often in the male. As yet we have not observed the disappearance of a high pulse pressure as aortic valve obstruction increases in a patient with 'free' aortic regurgitation. In time, we may observe such an occurrence.

Certain sex differences in the natural history of rheumatic fever and rheumatic heart disease are being observed. There is a strong female preponderance in this series. Despite this, differences are becoming evident and seem significant. Prior to adolescence, rheumatic fever and heart disease seem to be equally severe and fatal in the two sexes. Further, severe rheumatic heart disease (with considerable cardiac enlargement) in the adult male presents a poor prognosis as it does also in the female. However, the adult male without severe degrees of cardiac enlargement seems to have less trouble in adult life than does the female. Recurrent rheumatic fever appears to occur oftener in the adult female than in the adult male. 'Pure' chorea seems less important in relation to the development of rheumatic heart disease in the male than in the female.

So-called 'pure' mitral stenosis occurs less frequently in the male than the female. As these differences are being studied at present, actual data must be presented later. However, such differences seem important and need careful evaluation and explanation.

The frequency of hemolytic streptococcal infections appears to be an important factor in the outcome of rheumatic fever and heart disease patients. In time of war rheumatic fever occurs as an epidemic disease during mobilization, associated with hemolytic streptococcal infections. In peace time initial and recurrent rheumatic fever seems to occur oftener in the female than the male. Not only does the female experience the mechanical problem of bearing children, but also she must care for these children in their early years. This means a second period of frequent exposure to epidemic hemolytic streptococcal infections. Exposure to their children's respiratory illnesses may be a significant feature in the natural history of rheumatic fever in the female adult. Whether this explains the rheumatic fever differences between the adult male and female remains to be seen. These are suggestive but ill-defined inferences that hormonal factors may possibly play some role. Further study is needed.

It may be well to summarize briefly the experience in this series of patients with regard to prognosis. Since 20% of these patients died prior to adolescence, certain prognosis is inadvisable prior to this time. At an early age, death is customary as the result of severe, recurrent rheumatic fever, associated with a preceding hemolytic streptococcal infection. The war experience did much to direct attention to the fact that rheumatic fever occurs in a varying percentage of those individuals having epidemic hemolytic streptococcal infection. We may assume that rheumatic fever in a population clearly indicates epidemic hemolytic streptococcal infections in the community. This sharply focuses attention on environmental, as opposed to heredity factors. It also becomes apparent that in order to make progress against this disease we must orga-

nize programs of care, which would tend to minimize the exposure of rheumatic fever individuals and their families to such epidemic factors. This is not easy to accomplish and few satisfactory community programs are at present in operation in the United States. The methods for evaluating the results of such a program have not been clearly defined up to the present. Sulfa drug prophylaxis has been a tremendous impetus in relation to the protection of patients from such infections. This has many opponents as well as many enthusiastic proponents. It seems obvious that there is much about sulfa prophylaxis which we must learn and the final outcome of such prevention measures is unknown. However, the development of further prophylaxis studies are urgently needed. Such measures may not be the final solution to the problem, but a number of excellent observers feel that protection can be given in this manner.

A possible exception to the statement concerning the undesirability of making a prognosis prior to adolescence, is found in the patient with chorea. Individuals who define themselves as 'pure' chorea do better than those with other manifestations of rheumatic fever. This is true of both sexes, but especially of the male, most of whom reach adult life without evident heart disease.

Once full growth has been attained, the prognostic feature of most importance seems to be the size of the heart. Individuals with considerable degrees of cardiac enlargement rarely live beyond mid-life. The story of this group of patients is well known. Differences in the two sexes have been mentioned and may offer leads for further study. Also, we must be alert to evaluate the presence of active rheumatic fever in the adult, especially in the female exposed to frequent respiratory infections of young children. I should like to make an appeal to physicians to interest themselves in long term observations and supervision of patients with such a chronic, repetitive disease as rheumatic fever. Funds have not been available for observers to study the varying factors which

seem to influence the progress of this disease. Recently, in order to stimulate interest in rheumatic fever there has been formed an American Council on Rheumatic Fever, under the auspices of the American Heart Association. This Council is composed of delegates from leading national professional organizations and individuals who have spent some years working in the problem. The Council is just beginning to function. The stimulus for the development of such a Council came from the state programs now operative under the auspices of the Children's Bureau of the Federal Security Agency, and the experience with rheumatic fever as an epidemic disease in the Armed Forces. A conference, attended by a large number of interested representatives of public and voluntary organizations, was held to discuss what might be done about the problem. Without dissenting voice the conference went on record as favoring the development of a Council under the auspices of the American Heart Association. The Council has several objectives: The first is to stimulate the development of community programs. There are only limited programs throughout the country at the present time, facilities are limited and there has been relatively little co-ordinated planning for this important health problem by voluntary and public agencies. The Council hopes to stimulate the development of further planned programs of care at both voluntary and public levels. The second purpose is to make the public aware of the problem. In addition, professional education is to be made available. Finally there is the question of research. There are a number of suggestions which need extensive and expensive investigation. Some of these are fundamental in nature, while others are more developmental, particularly with regard to evaluating the results of treatment. There is an urgent need and a great opportunity to increase our knowledge concerning this soluble problem.

As a group you are aware of this problem and the need for further knowledge. Therefore, I express to you a strong hope that as individuals you seize any opportunity to help in your local communities or in your professional life by supporting

any activity which will aid in the ultimate accomplishment of the purposes of the American Council on Rheumatic Fever. If these purposes can be partially achieved in the next few years, a much neglected health field will have been explored with doubtless benefit to sufferers from rheumatic fever and heart disease.

In closing, I should like to congratulate you upon the development of the Life Insurance Medical Research Fund as discussed before you this morning by Dr. Francis R. Dieuaide. This is a great advance. We can apply only that which we know and understand. Your help will not only be desirable but essential, if the Research Fund is to accomplish its worthy purpose. Here is an opportunity, if funds are significantly increased, to circumvent many of the ills of the grant-in-aid system of research financing. Along with many investigators, Doctor Dieuaide is well aware of these difficulties. Stable, long-term financing of full time investigators is a great need in medical research. May the Life Insurance Medical Research Fund result in much needed knowledge in the field of cardiovascular disease.

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PRESIDENT STREIGHT—Thank you, Dr. Jones, for this very interesting discussion. The question is now open for discussion from the floor.

A MEMBER—Could you tell us how long after rheumatic fever begins before there is a detectable increase in heart size apparent?

DR. JONES—I think it may come overnight in a very ill case. Usually it is slower, but striking and surprising changes occur, and at the end of the tenth or the twelfth day of fatal rheumatic fever the heart size may increase obviously in five or six hours as the fever comes down.

SAME MEMBER—Would that be true of permanent heart enlargement?

DR. JONES—I think it is entirely a dilation. I doubt if you would ever have a disappearance of heart disease, a recovery immediately, although in a period of five or six years it might very well be.

DR. LANGNER—What is the present status of Coburn's intensive salicylate therapy?

DR. JONES—I think I have read four or five papers a day on that, the majority of which have been against the intravenous salicylate suggested by Coburn. One or two have been partially supportive. Certainly, our experience—and I believe it is nearly everyone's experience—has been that intravenous salicylate in large doses may no longer be justified. However, we were impressed with the fact that we observed a reasonable amount of decrease of the severity of rheumatic fever under salicylate therapy with high blood levels. I believe the majority of individuals ultimately go back to treating rheumatic fever with salicylates because of its effect on symptomatology and not with the idea of curtailing the duration of the rheumatic fever or preventing heart disease.

DR. BERTHOLD T. D. SCHWARZ—In determining the presence of heart impairment due to rheumatic fever, I believe you have included in that study the presence of a prolonged interval as the only sign, perhaps. My question is, have you observed much variability in the duration of the P-R inter-

val—for instance, an individual showing a P-R interval between .23 and .25 and at other times it may be only .20, or less?

DR. JONES—I didn't.

DR. SCHWARZ—The duration of the P-R interval—does it change in your experience?

DR. JONES—Of course, it will change very quickly in many instances. I think, however, the question of change has been greatly overrated, and that, certainly, in chronic rheumatic fevers the prolongations are markedly fixed for long periods of time. I may say that in no single instance in the thousand patients I mentioned was the P-R interval alone considered sufficient evidence to indicate heart disease. I believe that in a small number of individuals unquestionably it is probable that prolongation of the conduction time is supracardiac rather than indicative of active carditis.

DR. RICHARD S. GUBNER—Dr. Jones, your study was carried out on a group who received unusually protracted care. I wonder what is the life cycle of rheumatic fever, in your opinion, in the general population, such as we are called upon to evaluate in insurance work where protracted cardiac treatment is not given as a general rule, nor good follow-up?

DR. JONES—I can't answer that. I wish I could. I may say I have never felt that I had altered the course of rheumatic fever, except, perhaps, for the individuals with protracted failure whom I have kept alive by helping them mechanically until the disease subsided to the point of again having a competent heart. Dr. Bland was particularly interested in the question of getting a 'pure' series together, and the variations and variables are so terrific that we have never succeeded. I doubt very seriously if the prolonged care is the important feature. I say that very advisedly because long ago I gave up the idea that a place like the House of the Good Samaritan justified its existence simply because it kept people in bed who didn't develop heart disease. I think what it does is that

it protects them from exposure to factors that are known to affect a disease such as rheumatic affections. We have now a complete service and a fresh atmosphere. If patients get into our hospital and do not have more pain after they get in, we expect them to walk out. I am not certain but probably the most significant reason was the feature of separate beds. We instituted it; we insisted on it, no matter what the home surroundings were, and the social agencies and the welfare agencies got their heads together and are supplying that individual with a bed by himself. In some of our worst cases we had three or four children sleeping in one bed, and sometimes two or three of those had rheumatic fever. They did not have a chance to escape the factors of infection that are at least associated with that disease.

DR. ARTHUR E. PARKS—Dr. Jones, one of the most important matters that we have to decide, in the selection of risks in our work, is the question of mitral stenosis and aortic valve involvement. Up until now, we have had to take a very serious view of these conditions. I have been led to believe from your paper that if the heart size is normal we may be able to take a more lenient view of these impairments, particularly in the case of women who have mitral stenosis and in the case of men who have aortic involvement. Would it be your opinion that we should revise our opinion toward these impairments for the purpose of selecting them as risks?

DR. JONES—I prefer not to answer that. In twenty years, however, I don't think there is any question but that those murmurs are indicative that those lesions alone have had far too serious significance paid to them. I think that the heart size is infinitely more important than either of those two lesions, although many of our males as they get to be old men get mitral stenosis. I don't know but I would state that very few, certainly the large majority of males with mitral stenosis, without much enlargement, will do extremely well. I think in the case of mitral stenosis that 33 are 'pure' mitral stenosis out of a thousand, but if a person has good cardiac reserve

and little or no abnormality in the size of the heart and doesn't have aortic regurgitation, I would be inclined to think he was a pretty good subject. His living conditions would probably be as important as his heart findings — how closely he is exposed to his own or a number of other small children.

For instance, we lost one of our best physicians in Boston a few years ago. He had a tremendous amount of rheumatic heart disease, but he carried on for a great many years, and then suddenly he went smash, with aortic regurgitation. Why, his necktie used to bob up and down when he spoke loudly. He insisted on taking care of the boys in a prep school, to which he was greatly devoted because he was a graduate, and he died, as a result, of typical recurrence of rheumatic fever which he had contracted by taking care of an epidemic of streptococcic throats in that school. I think there isn't anyone here who would have paid any attention to the question of curing that man if they had gone on the basis of ordinary heart findings, and yet for more than twenty years he carried on magnificently and then got nipped, not by his mechanics but by a recurrence of the disease.

DR. ERNEST J. DEWEES — May I ask Dr. Jones whether in his experience he has seen an increase in coronary disease among the rheumatic cases that have recovered?

DR. JONES — We have been very much interested in that because of the fact that some of our patients have coronary pain, sometimes very severe coronary pain, and of course, there is a well-known work of Karsner in relation to rheumatic fever and coronary disease, but in our group we have not observed it as yet. Of course, it may turn out to be true when they get older but up to the present time certainly, there has been no increase in coronary disease since the '30's in our hospital.

PRESIDENT STREIGHT — Our next paper is on "Exploratory Electrocardiograms" and will be given by Dr. Jan Nyboer, Assistant Medical Director of the Connecticut Mutual Life Insurance Company. Dr. Nyboer!

EXPLORATORY ELECTROCARDIOGRAMS
EXTREMITY, PRECORDIAL, ESOPHAGEAL
AND
DISCUSSION OF THE Q_3 ELECTROCARDIOGRAM

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EXPLORATORY ELECTROCARDIOGRAMS

The interest and usefulness of the exploratory leads in electrocardiography are steadily increasing in clinical, public health, and industrial medicine. The introduction of mechanical recording will make the technique more convincing and useful; however, the principles (1,2,3,4,18a,18b,21,42), and results of exploration have been well established before this advent.

The purpose of exploring is to discover and amplify local processes (2,3,4,21) in the heart, such as infarction and ectopic foci of idiopathic rhythms not easily demonstrated in the standard potentials. Arrangements to tap the unipolar potentials of the extremities are easily accomplished by the methods of Wilson, et al. (12,13) and Goldberger (42). The form of indirect potentials from the anterior myocardium, using any one of several indifferent electrode connections (15), is now quite well understood. There has also been some investigation of the retrocardiac potentials from the surface of the body (45,49); however, the best and nearest approach to this region is made by exploring the potentials in the esophagus (5,6,7,8,33) at well-defined anatomical levels. The principal purpose of this paper is to correlate and demonstrate the form of these various regional electrocardiograms in conditions which are established from the history and findings in each case.

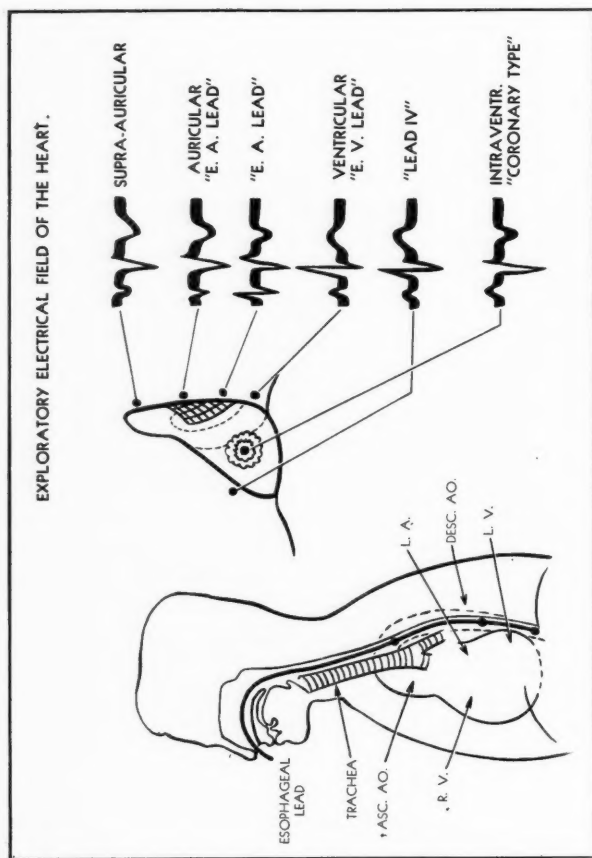
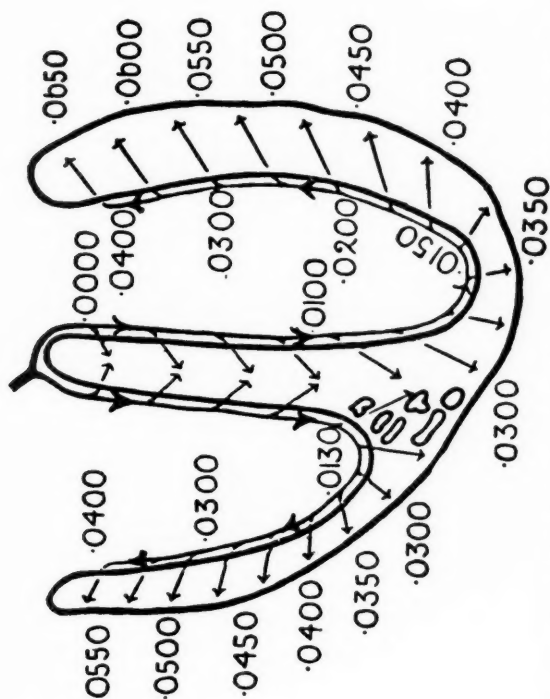


Fig. 1A

Diagram showing the esophageal electrode in situ. Diagram of the general form of the exploratory potentials, anterior, inside, and posterior to the heart.



A diagram of the human heart in section, representing the directions in which the excitation wave spreads in the human ventricle and the time in seconds at which, after its commencement in the ventricle, the wave first reaches various regions of the ventricle. (After Lewis.)

Fig. 1B

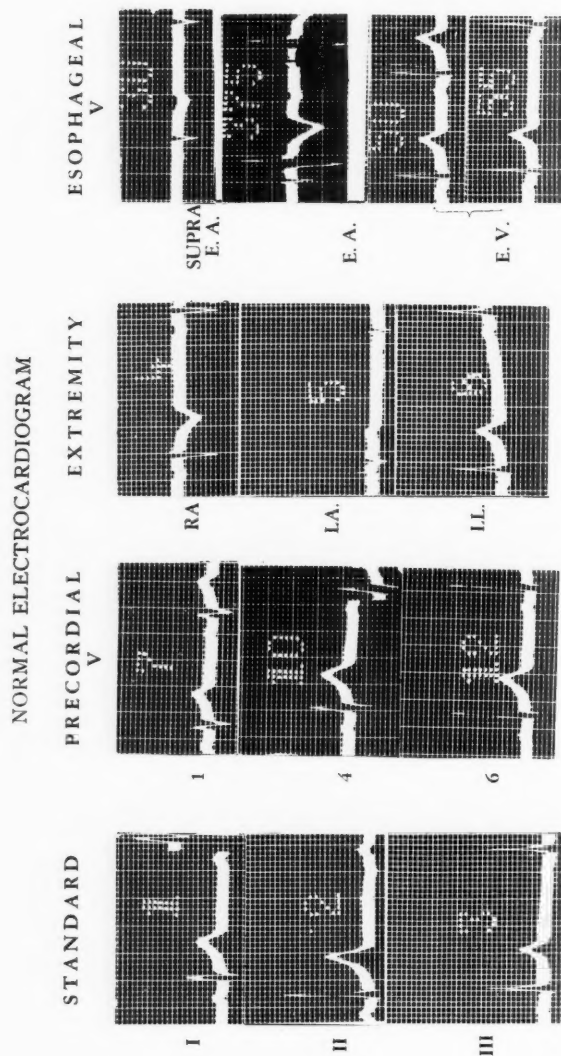


Fig. 2A

Electrocardiogram from normal male subject, age 22.

Figure 1a illustrates the method of introducing an electrode at the end of a No. 12 Levine rubber catheter via the nasopharynx and esophagus to the stomach. In this manner indirect potentials can be recorded from the left ventricle, left auricle, and aortic regions. The technique is identical to that of passing other enteric tubes. Topical anesthesia of the oral pharynx is imperative in irritable throats. Exploration (8) is usually made in 2.5-centimeter steps from a level of 60 centimeters to 25 centimeters from the nares.

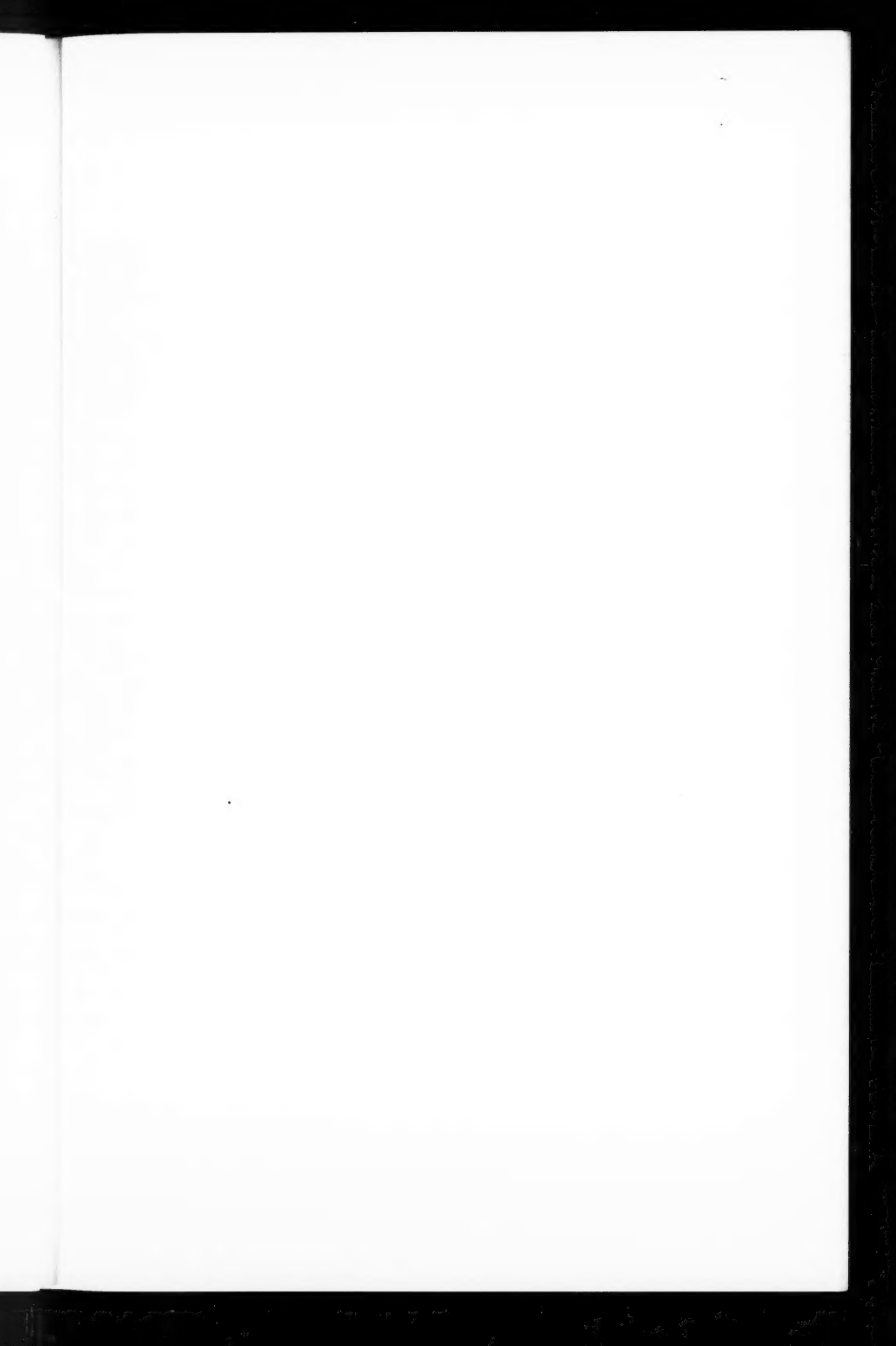
On examination, the graphs from the various anatomic levels are identified physiologically by the form of the auricular P-wave (Fig. 1a, 2a). A Wilson's central terminal is recommended as the indifferent electrode. The potentials are recorded so that an electropositive deflection is directed upward. The *supra-auricular level* is identified by a smooth or notched inverted P-wave. During the chief P-wave deflection the action current is traveling away from the electrode. The auricular level (E.A.) is identified by sharply diphasic or polyphasic P-waves. The form varies physiologically in variable anatomic relation to the sinus pacemaker. The upper region is chiefly electronegative, and the lower region is chiefly electropositive. These deflections are usually much larger than those of the standard leads. The sharp plus-minus deflection (similar to the RS complex) is known as the auricular intrinsic deflection of Lewis (18a) and is absent in supra-auricular and ventricular potentials. The P-wave at the ventricular level (E.V.) is smooth or notched and upright anteriorly and posteriorly. During its chief deflection the action current moves toward the electrode position.

The form of QRS and T is also typical at each level and over each ventricular chamber, as illustrated (Fig. 1a, 2a). In brief, at the supra-auricular and auricular (E.A.) level, QRS is chiefly electronegative, as the action current is traveling away from the electrode. At left ventricular levels anteriorly (IV) and posteriorly (E.V.), the QRS-T complexes are usually similar and predominantly electropositive and, therefore,

upright, as the action current is moving toward the electrode near the epicardium. Depolarization of ventricular muscle usually proceeds from the endocardium (Fig. 1b) and moves toward the outer surface. The *onset of the RS* is at the moment the area under the electrode becomes electronegative. This is known as the *ventricular intrinsic deflection*, and it occurs earlier in the QRS cycle over the right ventricle than over the left ventricle, as the walls are, respectively, thin and thick. The process of activation, therefore, varies in time. Normally, R is short and S is long over the right, and R is long and S is short over the left ventricle. All portions except the RS, Q or QS inscriptions are extrinsic movements of the action current in direct or indirect leads over the ventricles.

The form of *potentials in the ventricular cavity* is also predetermined, and usually these are the inverted form of the epicardial potentials. As the endocardium is electronegative, the moment the signal is distributed by the Purkinje network, the action current immediately travels away from any electrode in this position. The P-wave is electropositive, while the QRS and T are chiefly electronegative. This was demonstrated on frog and cat hearts by MacCleod (19), on dog hearts by Wilson (21,26), et al., and recently on human hearts by Hecht (34). The electrical potentials over large areas of myocardial infarction are similar to the intraventricular potentials, as the polarizing process of the wall has been transplanted by one of volume conduction, such as takes place in metal conductors.

In regard to *unipolar extremity leads*, the contributions of Wilson (13), Hecht (16), Goldberger (45), Myers (35), and Lyle (36) are particularly helpful. The left leg potential in differentiating physiological and pathological Q-waves is promising. In general, the P-wave, QRS, and T-wave are principally negative in the right arm potential in normal cases, whereas the left leg potentials are principally electropositive. The left arm potential is nearly iso-electric when the electrical axis is normal; however, a shift to the right produces electro-



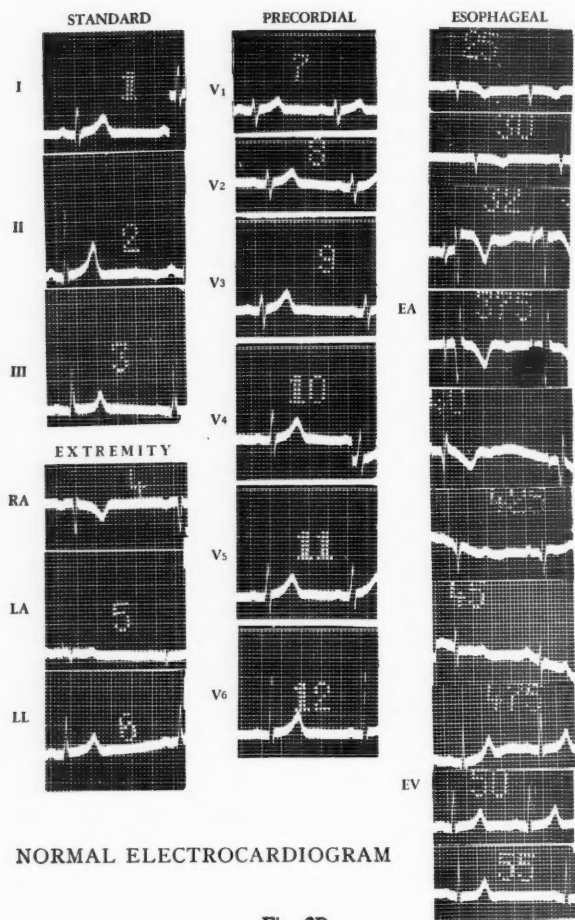


Fig. 2B

This is a detailed observation of the preceding normal study and reveals the usual transitional patterns from right to left ventricle, anteriorly, and from lower to higher regions in the esophagus in relation to ventricle, auricle, and regions above this, posteriorly.

negative patterns, and a shift to the left, electropositive patterns. Wilson (46) reported to this convention the usefulness of extremity potentials in association with the standard and precordial leads in predicting and differentiating whether the axis was affected by hypertrophy or merely rotational shift in the anatomic axis of the heart.

In medical underwriting we, too, are confronted with the problem of becoming more familiar with all types of exploratory leads. How else can we become familiar unless we constantly review our subject, our typical cases, and try to understand the physiological background of this recognized diagnostic tool in heart disease? I trust the following cases will assist each of us to formulate a more rational concept of the electrical field of the heart. For brevity's sake, we will delete some subjects, but these will appear in the final monograph. Precordial and esophageal electrocardiograms are selected and illustrated from the various levels indicated; however, the discussion is based on six precordial and about ten esophageal exploratory points, three unipolar extremity, and the conventional standard potentials.

The Detailed Normal Study and Considerations Using Standard, Unipolar Extremity, Precordial, and Esophageal Leads.

The graphs (Fig. 2b) were obtained from a normal male subject, aged 22. These were previously illustrated in less detail using one selected lead from the region of the precordium and two from the esophageal region of the left ventricle and auricle. (See Fig. 2a.)

The standard leads ($\frac{N}{I}$ sensitivity) are within normal limits of the Table I elaborated by Wilson and Nyboer (9). They show a normal electrical axis of approximately 60° , a normal PR interval of 0.16 seconds, and a normal QRS interval of 0.08 seconds.

The unipolar extremity leads (12), were taken as plus-minus exploratory potentials ($\frac{N}{I}$ sensitivity) using Wilson's central

TABLE I
THE SIZE OF THE VENTRICULAR DEFLECTIONS IN THE STANDARD AND
SPECIAL LEADS (MEASUREMENTS GIVEN IN TENTHS OF A MILLIVOLT)

LEAD	Q				R				S				T				RS			
	MINIMUM	MAXIMUM	MEAN	STANDARD DEVIATION	MINIMUM	MAXIMUM	MEAN	STANDARD DEVIATION	MINIMUM	MAXIMUM	MEAN	STANDARD DEVIATION	MINIMUM	MAXIMUM	MEAN	STANDARD DEVIATION	MINIMUM	MAXIMUM	MEAN	STANDARD DEVIATION
I	0	1.5	0.33	0.45	1.5	19.4	6.81	3.27	0	5.0	1.67	1.35	1.0	5.5	2.21	1.05	3.0	20.6	8.63	3.20
II	0	2.0	0.43	0.61	4.0	22.0	11.99	4.39	0	8.0	1.53	1.92	1.0	6.0	2.97	1.10	8.0	23.0	13.76	3.72
III	0	2.0	0.54	0.60	1.2	18.0	8.50	4.33	0	13.0	1.27	2.40	0	3.0	1.49	0.75	3.2	18.0	10.12	4.10
RA	0	7.6	2.81	2.68	0	3.0	0.76	0.65	0	10.5	2.56	3.41	-3.3	-0.8	-1.66	0.61	3.5	11.7	6.30	1.82
LA	0	1.5	0.21	0.42	0	7.0	1.13	1.34	0	7.0	2.00	1.60	-1.0	1.0	0.05	0.41	0.5	8.5	3.27	1.85
LL	0	1.2	0.29	0.37	0	13.0	6.68	2.81	0	6.5	0.80	1.28	0.2	2.8	1.46	0.62	4.0	13.0	7.66	2.54
V ₁	0	0	0	0	1.0	9.6	4.16	2.33	3.4	24.0	11.05	5.03	-4.0	5.6	1.23	1.88	6.6	26.8	15.21	5.98
V ₂	0	0	0	0	4.0	20.8	9.05	3.68	3.0	38.8	16.23	7.30	2.4	11.0	6.22	1.90	15.0	46.0	25.27	7.47
V ₃	0	0.4	0.013	0.072	6.0	54.6	16.70	9.78	0	22.0	9.05	5.62	3.6	12.0	6.26	1.89	12.6	54.6	25.75	8.37
V ₄	0	3.0	0.37	0.68	12.2	46.0	22.31	7.10	0	16.0	5.32	4.13	2.4	11.0	5.66	1.84	18.0	51.6	27.63	6.71
V ₅	0	3.4	0.57	0.91	8.8	33.0	18.78	6.91	0	9.6	1.93	2.22	2.0	9.6	4.59	1.82	11.2	33.2	20.70	6.50
V ₆	0	0	0	0	2.0	12.8	5.81	2.49	0	16.2	6.09	4.78	0.2	5.2	2.55	1.11	5.6	24.2	11.91	4.33

The statistical study by Wilson and myself of 104 normal adult subjects shows some interesting facts. The absence of Q in the leads listed is quite normal. Q is never present normally on the extreme right precordium. Q is present normally on the left precordium and S, therefore, the earliest evidence of activity by exploring leads is on the left precordium. The Q is generally small in standard extremity, and precordial leads except in RA, where it is large. On the other hand, R is small in RA and may be large in LL. Like Q, S is large in RA and small in LL. Normally, S is prominent on the right, and R is prominent on the left precordium. T may be negative on the right but is always positive on the left. The picture in juvenile electrocardiograms is different with respect to T.

terminal as the indifferent electrode. Einthoven's law may be applied to these as well as to the standard leads. The sum total of the given electrical potentials of the three extremities, i.e., RA (the right arm potentials) plus LA (the left arm potential) plus LL (the left leg potential) is equal to zero. Mathematically this may be written $RA + LA + LL = 0$. On rearranging the equation $RA + LL = -LA$ and substituting values of RS (or QR) from the graph, it is readily seen why the left arm (LA, see graph) is nearly at isopotential in normal subjects with an electrical axis of about 60° . In this subject a bizarre QRS voltage is present in this lead. The comparatively large voltage of Lead II is self-explanatory, since it is the result of the difference in potentials between the foot and the right arm; in this case LL (+.86) minus RA (-.75) equals Lead II voltage (1.61 millivolts). A casual glance at QRS_2 in the graph confirms this observation, since it is approximately 1.65 millivolts. This law also governs the size and direction of the remaining deflections, i. e., P, T, and U.

The normal forms of QRS-T in the extremity potentials were initially described by Kossmann and Johnston (12), who studied thirty normal male subjects. Their results were summarized by Wilson before this convention in 1942 and are reproduced here (Table I). At the same time Wilson, et al., (46), reported a study of factors producing axis deviation in these leads. In general the chief deflection in QRS and the T-wave is electronegative and downward in RA, while in LL the chief deflection in QRS and the T-wave is electropositive and directed upward in normal subjects. Lead LA shows considerable variation with slight or moderate shift in electrical axis; the chief deflection in QRS is usually electropositive in left electrical axis, and it is usually electronegative in right axis deviation; the T-wave may be iso-electric, diphasic, electropositive, or electronegative in normal cases; this is confirmed by the normal table of these potentials. No known data is available on the U-wave of these electrocardiograms; how-

ever, it is observed as inverted in leads opposite the auricle and usually upright in precordial and esophageal ventricular leads.

The normal forms of the P-wave in the extremity potentials are of interest since they are often compared with exploratory potentials elsewhere. In RA it is inverted and electronegative; in LA it is usually diphasic or nearly iso-electric; and in LL it is upright and electropositive normally. When dextrocardia is present and the pacemaker of the action current is in the sinus node, the P-wave in the RA lead becomes upright and electropositive. If the P-wave in Lead I is difficult to differentiate, the decision of normality or abnormality regarding the pacemaker may be made by this accessory lead (RA).

The precordial potentials were taken as plus-minus exploratory leads ($\frac{N}{2}$ string sensitivity) using Wilson's central terminal as the indifferent electrode. The summary of the voltages found in thirty normal male subjects with serial precordial leads was also originally reported by Kossmann and Johnston (12). Their results are also combined in Table I.

The various potentials of the precordium across the region of the ventricles are defined as follows: V_1 is obtained from the right sternal border and fourth intercostal space or lower; V_2 from the left sternal border and fourth intercostal space or lower; V_3 over the fifth rib and halfway to the mid-clavicular line; V_4 over the fifth intercostal space, mid-clavicular line; V_5 over the sixth intercostal space and anterior axillary line; V_6 over the seventh intercostal space and left mid-axillary line. These various points may be explored in connection with other indifferent electrodes and designated when it is CF (chest-foot), CR (chest-right arm), CB (chest-back), or CL (chest-left arm) connection (15).

In normals it is seen that R becomes progressively larger as we pass from right to left, reaching a maximum voltage at points 4 or 5 and after this usually diminishing. The *S wave* is usually maximum at point 2 and becomes progressively

smaller on transition to the left. It may often disappear at point 4 or 5. A small Q may be found normally at points 4, 5, or 6. T is usually largest at point 2 or 3 and becomes smaller to the right and left of this position. RS is referred to as the ventricular intrinsic wave; it begins rather early over points 1 and 2 and occurs usually in the first one-quarter or two-hundredths of a second after the beginning of the normal QRS interval; it begins later over points 4, 5, and 6, and is usually mid-way in the QRS interval or is about four-hundredths of a second in time. The position of the beginning of the intrinsic wave varies with hypertrophy (16), bundle branch block (12), and myocardial infarction (10), particularly of the anterior wall.

The Normal Study of the Esophageal Lead (7,8): (Fig. 2b) An electrocardiogram is made from an electrode tip placed in the lower end of the esophagus or cardiac end of the stomach (55 to 60 cm. from the nares) and then subsequently at 2.5 cm. steps above the preceding level until a region 25 to 30 cm. from the nares is reached. The central terminal of Wilson is again used as the indifferent electrode. An electropositive potential deflects the string upward, and an electronegative one deflects it downward.

Opposite the ventricle (55 to 45.0 cm.) the P-wave is usually smooth and upright, closely resembling P_1 and P_2 in general form. The QRS complex resembles that obtained from points 4 to 6 on the precordium. The T-wave is upright and resembles those points from 4 to 6. The Q-wave is never large normally, and if it exceeds 0.3 to 0.4 mv. or is greater than 20% of the R-wave in this same lead, it is probably abnormal in magnitude. The RS deflection is the intrinsic wave in this region.

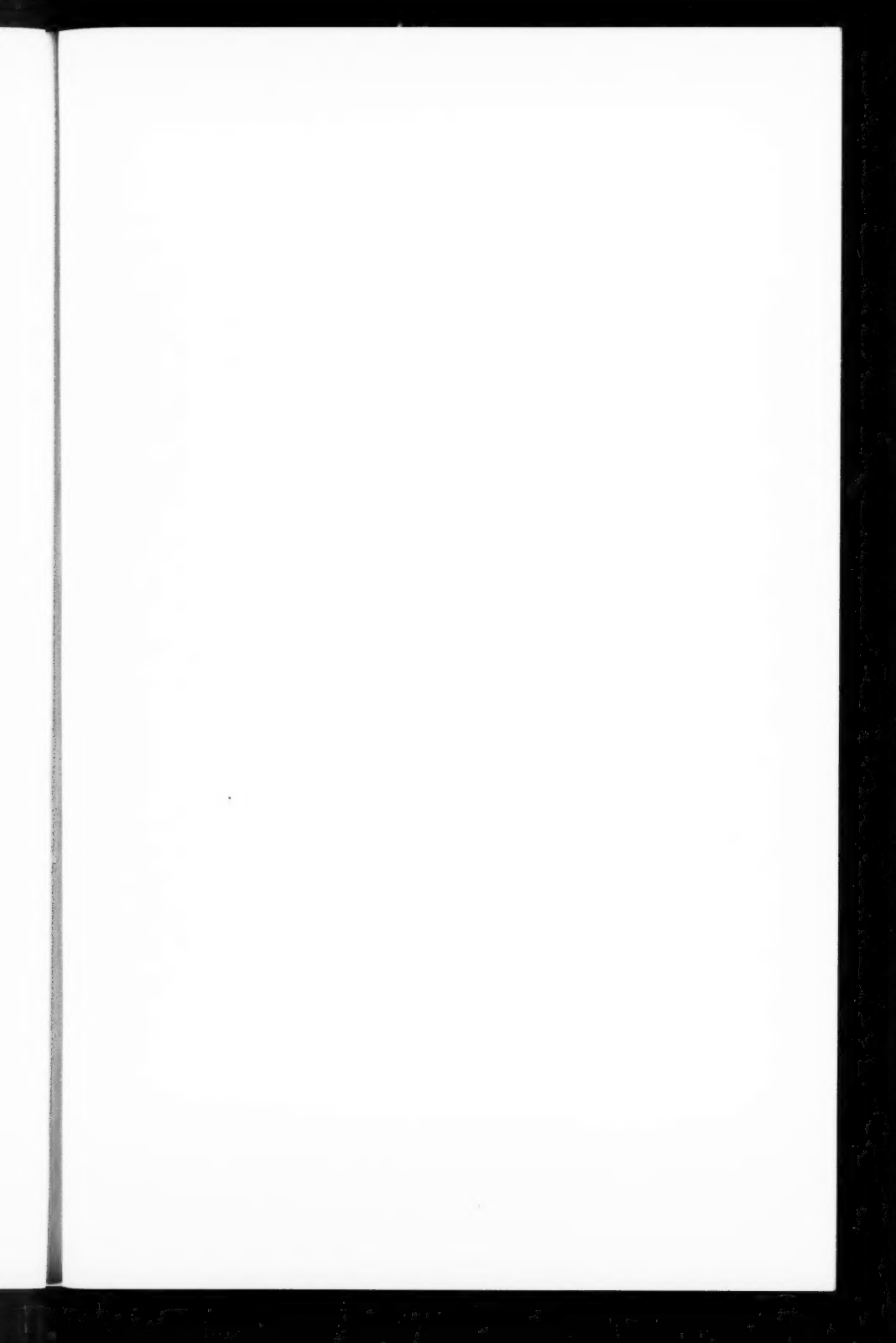
At the border of the auricle and ventricle (42.5 cm.) the electrocardiogram often takes on characteristics of both auricular and ventricular leads and, therefore, is difficult to define with respect to the individual deflections.

Opposite the auricle (40 to 32.5 cm.) diphasic or polyphasic P-waves are present at several positions. The sharp downward movement in the P-wave is the so-called auricular intrinsic wave of Lewis (18). This wave is not found in the P-waves of the standard leads, nor in any exploratory leads directly or indirectly over ventricular muscle or of the extremities. This type of P-wave is a physiological index of the anatomical position of the electrode adjacent to the auricle (7). The major deflection of the P-wave of the E.A. lead is inscribed above, equally, or below the isopotential line, depending upon the position of the lead in relation to lower, middle, or upper fraction of the left auricle. Often a depressed segment is noted following the P-wave. This is evidence of the T-wave of the auricular deflection. It is sometimes responsible for occasional slurring of the QRS deflection when merging with it (19). The QRS complex usually has a deep Q followed by an R spike of variable size. The RS-T segment is usually iso-electric and the T-wave is normally inverted in these leads.

Above the auricle the P-wave is smooth and inverted, resembling that found in the unipolar right arm potential (RA). The QRS and P-wave also follow the characteristics of this same lead.

U, the sixth wave of the electrocardiogram, is usually upright in esophageal ventricular leads and inverted in esophageal auricular leads. It is inverted in leads above the region of the auricle when this wave is present. It is no larger than the same wave when found in the precordial potentials (20).

In summary, the serial precordial potentials have a characteristic pattern over the right ventricle and also over the left ventricular region. The latter closely resemble esophageal ventricular electrocardiograms. The esophageal auricular leads are easily identified by the characteristic P-wave (usually diphasic) in normal subjects.



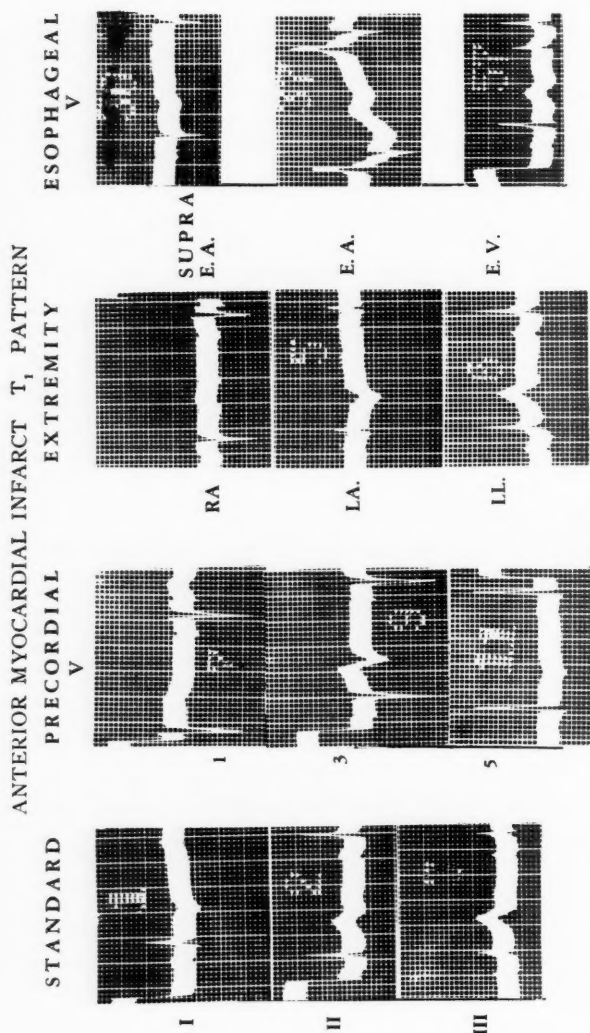


Fig. 3

An anterior myocardial lesion produced abnormally inverted T_1 , abnormal Q_4 , and normal EV patterns. Q_4 is deep because the entire thickness of the muscle under the electrode is probably infarcted. Extremity potentials are not greatly modified but R is present in RA and Q in LA and T is upright in RA.

Anterior Myocardial Infarction

B. B., a man aged 62, was admitted to the Bellevue Hospital on October 3, 1941. He complained of moderate precordial and back pain for about two days; however, vague precordial pain was present for two weeks before entry. Slight swelling of the ankles was noted in the evening over this same time. Dyspnea on exertion has been present for a long time. On physical examination the blood pressure was 100/58, the pulse rate 88, the heart sounds were fair, the rhythm was regular, no murmurs were heard, and moist rales were present at the posterior lung bases and higher. The initial electrocardiogram was equivocal. Later typical and progressive changes were found adequate to establish the diagnosis of anterior myocardial infarction. No digitalis was given at any time. There was no enlargement of the heart on a two-metre X-ray film.

The standard potentials (N_1 sensitivity) of October 17, 1941, (Fig. 3) show a low voltage tendency, a normal electrical axis, a small Q_1 , an iso-electric RS- T_1 segment, and inverted T_1 . Except for the bizarre QRS_3 and sharply upright T_3 , there are no other striking abnormalities in these leads. These findings are suggestive of anterior myocardial infarction.

The single extremity potentials ($\frac{2N}{I}$ sensitivity) show a low voltage, diphasic T in lead RA and sharply upright T-wave in lead LL as the major deviations from normal. A small Q-wave is also present in LA and a small initial R is present in RA. These combine to form a Q-wave in Lead I.

The precordial potentials ($\frac{N}{I}$ sensitivity) are normal at points 1 and 2 and abnormal at points 3, 4, and 5. The Q-wave is the only QRS deflection and is followed by a diphasic (plus-minus) T-wave at point 3. The QRS complexes are normal at points 4 and 5; however, the T-waves are abnormally inverted at these points. These findings are specific for anterior myocardial infarction. The central mass of infarction is probably below point 3.

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The esophageal potentials ($\frac{N}{T}$ sensitivity) are essentially normal above the auricle at point 30; however, opposite the auricle abnormal diphasic or upright T-waves are found at points 32.5, 37.5, and 45.0. Opposite the ventricle, the entire QRS-T is of the normal type at all points, including 50.0, 55.0, 57.5, and 60.0.

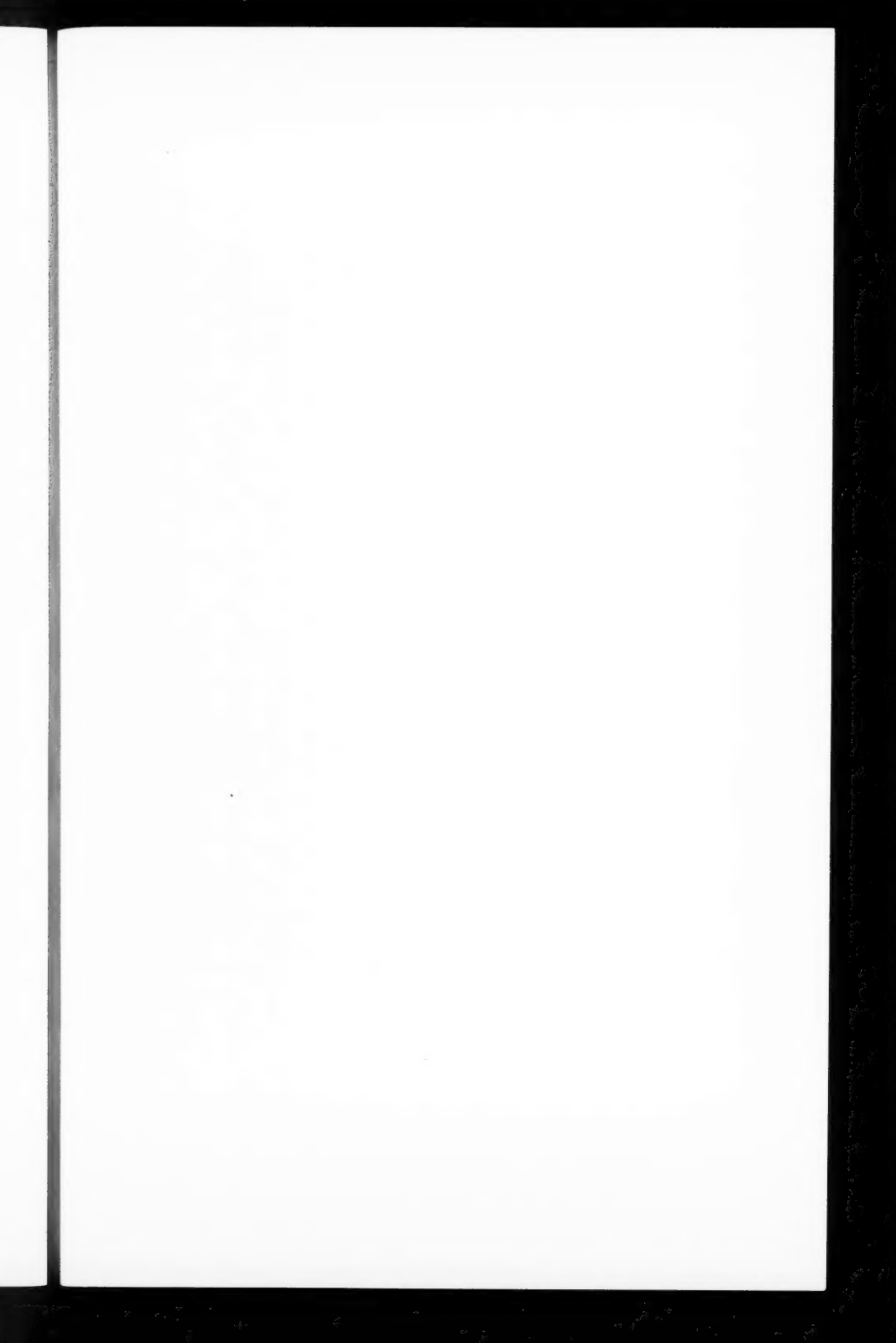
In summary, the standard leads are assisted by the precordial leads in establishing a diagnosis of anterior myocardial infarction. The esophageal ventricular leads were essentially negative in this study; this is consistent with the academic findings.

Incomplete Anterior Myocardial Infarction

W. B., a man aged 49, complained of intermittent stabbing pains for about two and one-half years, which started in the right wrist, radiated upward to the shoulder and then across the anterior chest to the substernal region. It was initially precipitated by exertion, but now occurs spontaneously three to four times at night. No relief followed administration of nitroglycerine. At present tolerance to exertion is limited to one-half flight of stairs, or walking a distance of one block.

In 1940 he had two admissions to Bellevue Hospital, where diagnoses of duodenal ulcer and arteriosclerotic heart disease were made. The positive findings at that time were: (1) The gastro-intestinal X-ray series showed a persistent deformity of the duodenal bulb; (2) The gall bladder study showed a normal outline but a sluggish evacuation after the fatty meal; (3) The electrocardiogram was suggestive of myocardial changes; (4) Partial relief of symptoms was obtained with intravenous administration of cobra venom.

The positive history and findings on this hospital admission were: (1) Inability to work for two and one-half years; (2) The aortic second sound was accentuated, and his blood pressure was 148/100 on admission and later 128/90; (3) The sedimentation rate was 35 and 42 mm. on two occasions; (4) Blood



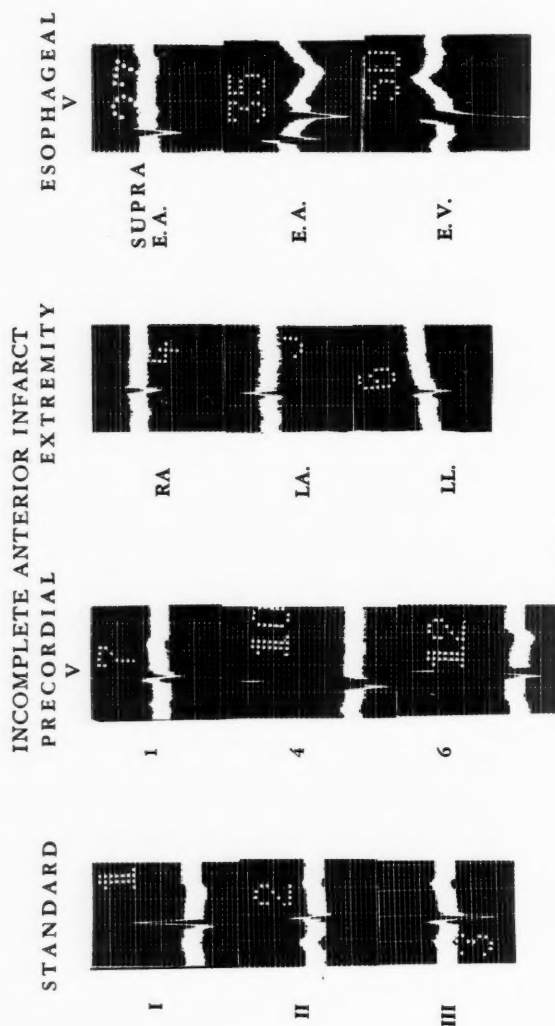


Fig. 4

Anterior myocardial infarction is present; however, judging by the depth of the Q waves in relationship to R on the precordium, the infarct does not involve the entire thickness of the heart wall underneath the electrode.

cholesterol and its esters were increased on two examinations; (5) A roentgenogram of the heart showed an aortic configuration and slight left ventricular enlargement; (6) An electrocardiogram showed moderate left axis deviation, abnormally inverted T_1 , low, upright T_2 , iso-electric T_4 , and borderline Q_4 of 0.4 to 0.5 millivolts. No conclusive evidence of localized myocardial damage was ascribed to these findings.

On April 19, 1941, a detailed electrocardiographic study was made, as shown in Fig. 4. *The standard potentials* ($\frac{N}{I}$ sensitivity) were similar to those described above. These findings, however, are non-specific for myocardial infarction of the anterior wall and might be found as a result of hypertrophy of the heart of any cause; therefore, the electrocardiograms are equivocal for myocardial infarction.

The single extremity potentials ($\frac{N}{I}$ sensitivity) are of no assistance in this case but are abnormal with respect to the T-waves in RA and LL, which are upright and iso-electric, respectively. Q is present in LA and initial R in RA. These combined to form a deeper Q in lead I.

The precordial potentials (about $\frac{N}{I}$ sensitivity) show normal findings at points 1, 2, and possibly point 3. At points 4, 5, and 6 the Q -waves are of the border-line type, measuring 0.5 millivolts or less. The T-wave is iso-electric at point 4 but inverted 0.25 millivolts at points 5 and 6, which findings are all abnormal. The RS-T segments are iso-electric throughout points 1 to 6. These findings again presuppose a change in the myocardium beneath the electrode at points 4, 5, and 6; however, they do not conclusively prove its local character unless it is absent from the posterior myocardium. In left ventricular hypertrophy a T-wave change also takes place in the esophageal ventricular region.

The esophageal potentials (about $\frac{N}{I}$ sensitivity) may be of assistance in this interpretation. Point 27.5 is not abnormal and is above the auricle. Points 35.0, 37.5, 40.0, and 42.5 are

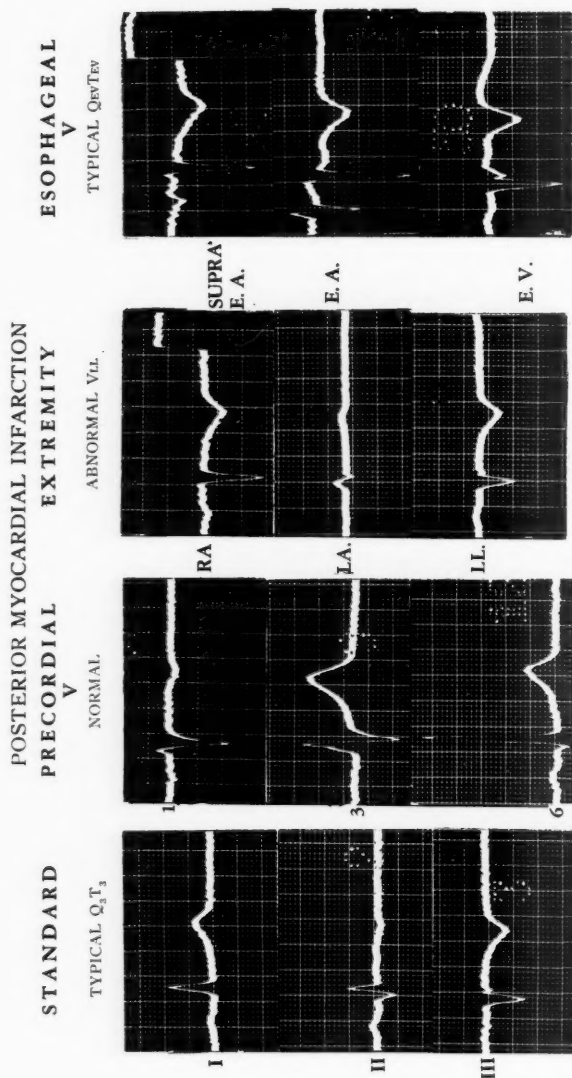


Fig. 5

A typical posterior myocardial infarction produced Q₃T₃ complexes and Q₂T₂ complexes in the standard leads. Lead IV is normal. Lead LL is abnormal and shows deep Q and inverted T. The esophageal ventricular lead is similar and localizes the lesion. (Note large size of P at the auricular level.)

Paper Speed 50 mm.

opposite the auricle and are possibly abnormal with respect to the T-waves, which are also modified by mechanical movement of the electrode during ventricular systole.

Points 45.0, 50.0, and 52.5 are in the region of the ventricle. These show an initial slurred R-wave of about 0.4 millivolts or less followed by RS, beginning mid-way in the QRS interval. This identifies its left ventricular origin, and, therefore, the QRS must be considered normal but modified, secondary to left axis deviation. The T-wave in each instance is normally upright but occasionally shows a mechanical artefact.

Thus, it appears evident that the changes in the exploratory electrocardiogram originate on the anterior myocardium of the left ventricular region. An infarct usually produces localized changes and in this case does not appear to involve the entire myocardial thickness beneath the electrode.

Typical Posterior Myocardial Infarction

J. E., a man aged 38, initially suffered from severe precordial pain following light physical work on August 7, 1940. This pain was not relieved by two hypodermic injections. He was then hospitalized, and a diagnosis of myocardial infarction was made by the routine electrocardiogram. His clinical course was satisfactory. He came to us for special study on January 16, 1941, about five months after his symptoms began. On physical examination, his blood pressure was 140/75, pulse rate 110, and there was no evidence of murmurs, abnormal tones, or enlargement of the heart. There were slight dyspnea, orthopnea, and anginal pain on moderate exertion.

The standard leads ($\frac{N}{I}$ sensitivity) show a Q_3T_3 type of electrocardiogram (Fig. 5) typical of a posterior myocardial infarction. It should be noted that Lead 1 is normal; Lead 2 is abnormal and has a Q of 0.3 millivolts, an iso-electric RS-T segment, and an inverted T-wave of 0.1 millivolt. Lead 3 shows an abnormal Q of 0.7 millivolts as the only QRS deflection, followed by a sharply inverted T-wave of 0.4 millivolts.

The PR interval equals 0.14 seconds. The QRS interval equals 0.08 seconds. (Time is recorded in 0.10- and 0.02-second intervals throughout this record only.)

The unipolar extremity potentials ($\frac{N}{I}$ sensitivity) are abnormal with respect to lead LL. This potential shows a deep Q-S complex followed by an inverted T-wave. The foot-potential often shows this deviation when there is infarction of the posterior-ventricular wall.

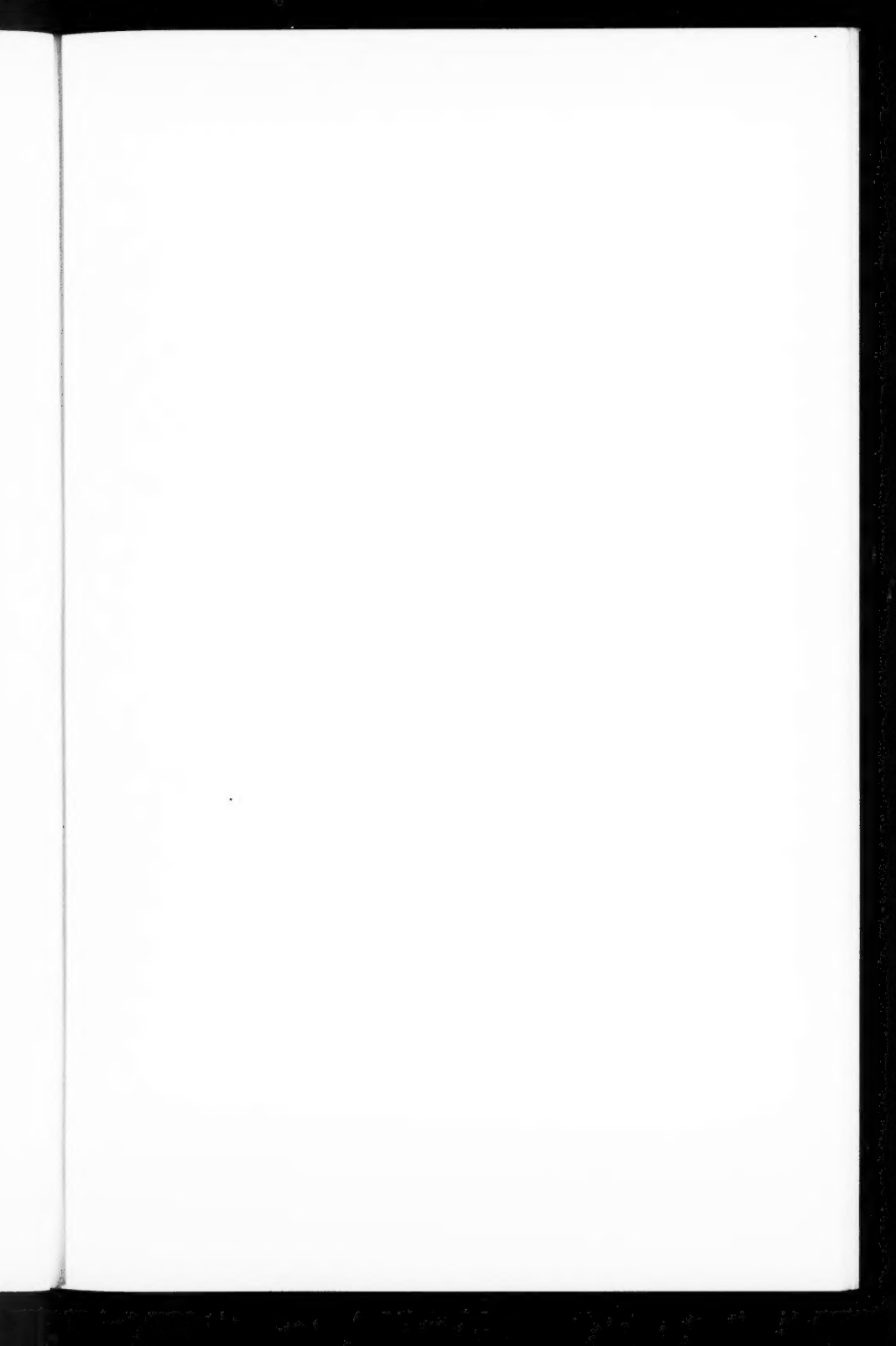
The unipolar extremity potentials of Fig. 5 and Fig. 7 are recorded incorrectly since the algebraic sum of these potentials at a given moment do not appear to be equal to zero. The form and magnitude of these potentials may be predicted from the standard leads by applying the following formula derived from Wilson's data.

$$\begin{aligned} RA &= -\frac{1}{3} (I + III) \\ LA &= +\frac{1}{3} (I - III) \\ LL &= +\frac{1}{3} (II + III) \end{aligned}$$

The augmented unipolar extremity potentials are 1.5 times these values.

The precordial tracings showed normal transitional changes from right sternal border (V_1) to left mid-axillary line (V_6) positions, all taken at normal string sensitivity. These indicate a normal anterior myocardium and closely resemble similar potentials taken from the normal subject (Fig. 2a and 2b).

The esophageal exploratory electrocardiograms ($\frac{N}{I}$ sensitivity) are normal opposite the auricle in tracings 35.0 to 42.5 cm. from the nares. Leads 5 cm. or more below the last-named level show an absence of an auricular intrinsic deflection. This is taken to indicate that the electrode is opposite the posterior portion of the left ventricle or below the diaphragmatic region of the heart when it is lowered into the stomach. In each of these tracings (45.0 to 60.0 cm.) an initial deep Q-wave of 1.3 millivolts (maximum) is the only QRS deflection present. It is followed by a deeply inverted T-wave. These



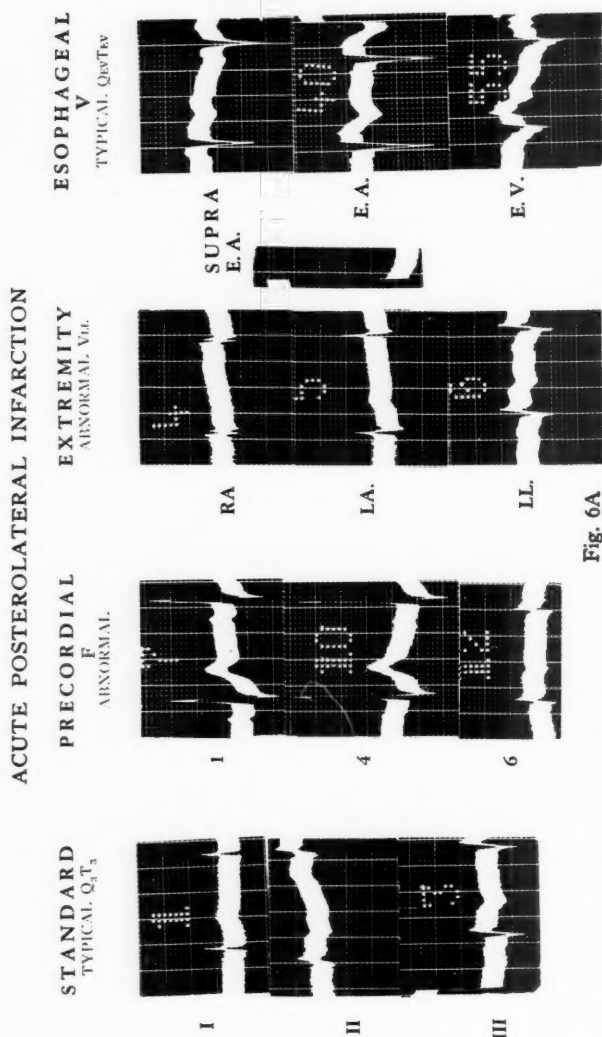


Fig. 6A

The deviations in RS-T segments are extremely interesting to follow in acute myocardial infarction. In this case of posterior lateral involvement, RS-T is elevated in leads II, III, LL, V₄, V₆, EV, and the 40 cm. lead. This signifies that the infarct is facing these areas, and subsequent electrocardiograms (Fig. 6B) reveal that T waves will be inverted in these same regions.

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are abnormal and localize the myocardial infarct in the posterior region of the left ventricle. Esophageal leads at 27.5 cm. and 30.0 cm. are taken above the auricle and are in the region of the arch of the aorta.

A study of the listed criteria for diagnosis of myocardial infarction summarizes the findings in the electrocardiogram in this disease. Thus, in this case a complete investigation of the electrical field of the ventricles has confirmed our impression obtained of this disease from the standard leads. The abnormal QRS and T in the E.V. lead are similar to the form of QRS and T taken directly from leads over the "central area" of infarctions produced experimentally in animals by Wilson (21).

Acute Stage of Posterolateral Myocardial Infarction

R. B., a male actor, age 64, two days prior to this investigation developed an acute, severe substernal and epigastric pain which was followed by vomiting while at a social function. He denied the use of alcohol at the time. Bystanders report he was in a cold sweat and almost fainted. Soon after this he was admitted to the hospital, where his pain was partially relieved by a hypodermic injection of morphine. On physical examination he showed signs of cardiac distress at rest. His blood pressure one month previously was 135/85; on admission, 134/94; and one day later, 98/62. The pulse rate was 78, the heart tones generally poor, a pulse alternans was present on the second day, the apex was 2 cm. to the left of the mid-clavicular line, and the temperature was 103° F. The rest of the findings were irrelevant to this study except for the (four-lead) electrocardiogram, which was typical of recent myocardial infarction.

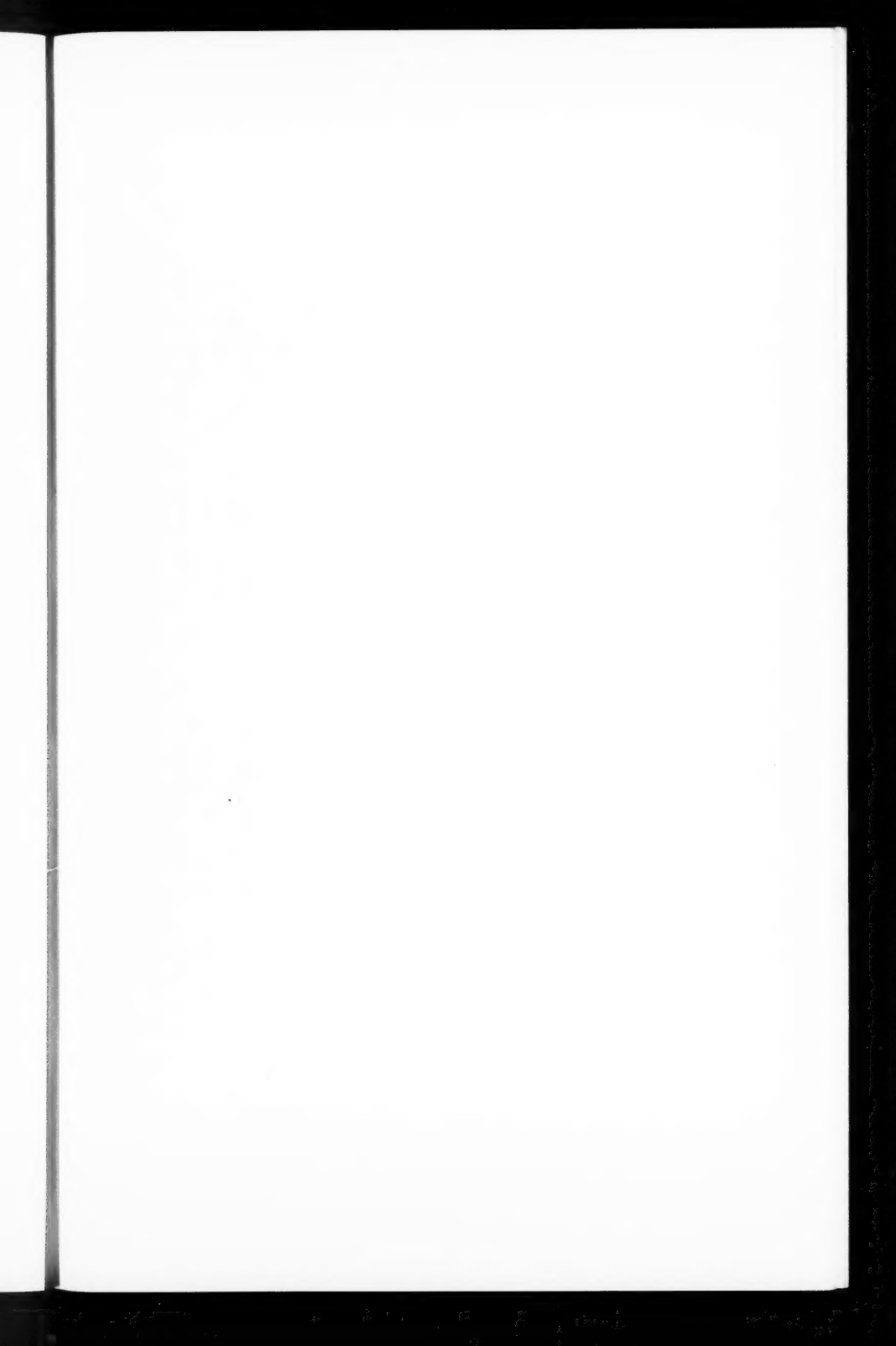
On October 7, 1941, (Fig. 6a) *the standard electrocardiogram* ($\frac{N}{T}$ sensitivity) shows low voltage P-QRS complexes in all leads. Lead I form is essentially normal. Lead II shows a Q-wave of 0.1 millivolts, an abnormally elevated RS-T seg-

ment of 0.3 millivolts, and a notched, upright T-wave. Lead III is abnormal throughout, showing a deep Q-wave of 0.4 millivolts, which is equivalent to the largest R-wave in Lead I. The RS-T segment is elevated 0.2 millivolts, and T is diphasic. These patterns are consistent with acute posterior myocardial infarction.

The extremity potentials ($2N$ sensitivity) show an abnormal, bizarre QRS in RA followed by a slightly depressed RS-T and iso-electric T-wave. Lead LA shows an abnormal, slightly depressed RS-T segment. Lead LL shows an abnormal Q-wave of 0.15 millivolts, equivalent to the tallest R in these leads. The QRS is followed by an abnormally elevated RS-T segment and diphasic T-wave. LL closely resembles Lead III and confirms the diagnosis of posterior myocardial infarction.

The precordial study ($\frac{N}{I}$ sensitivity) is abnormal in all positions examined. At points 1, 2, and 3 the R-wave appears exaggerated, and there is a depression of the RS-T segment. At points 4 and 5 the QRS complexes are normal; however, the RS-T segments are depressed. These findings at points 1 to 5 are consistent with an infarction directed on the opposite wall (posterior). At point 6 a bizarre, low voltage QRS complex initiated by a Q-wave is present. This is followed by an elevated RS-T segment and a diphasic T-wave. These last findings suggest a possible lesion of the lateral or apical region. They are similar to curves taken over the border-line of infarcts produced experimentally in dogs by Wilson, et al (21).

The esophageal electrocardiograms ($\frac{N}{I}$ sensitivity) in this case are of assistance in localizing the lesion suspected by the standard and unipolar extremity leads. Unfortunately, the P-waves are of abnormally low voltage in all the esophageal leads, as well as in the standard leads. This leads to difficulty in differentiating the curves at their respective levels; however, the tracing taken at 30 cm. is judged to be above the auricle; it is bizarre, low in voltage, and similar in many



SUBACUTE POSTEROLATERAL INFARCTION 24-X-41

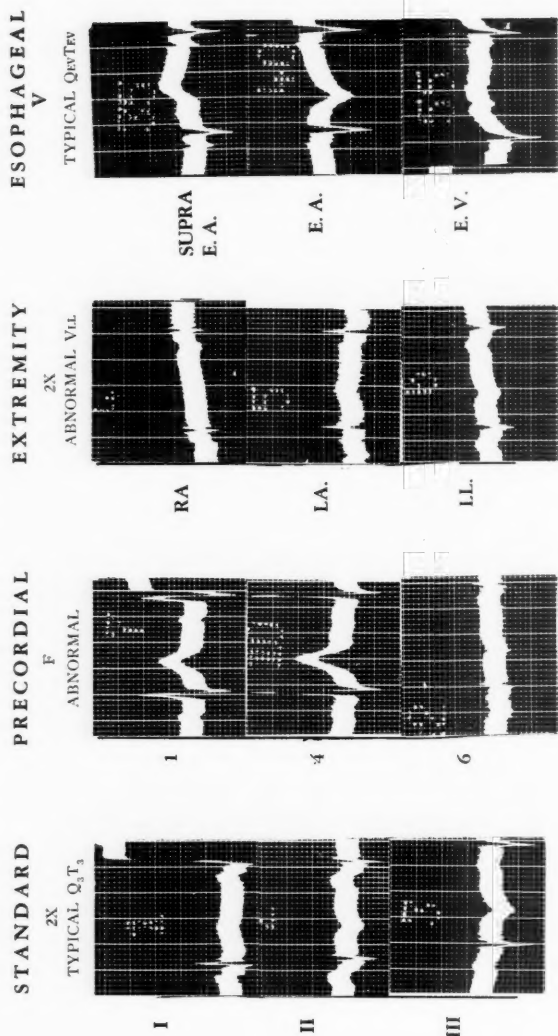


Fig. 6B

Progressive changes in the electrocardiogram 19 days following record 6A.

Exploratory Electrocardiograms 61

respects to RA. Curves labeled 35.0, 40.0, and 45.0 are questionably opposite the auricle. They deviate little from the normal form except for a slight elevation of the RS-T segment. Electrocardiograms designated 47.5, 52.5, 55.0, 57.5, 60.0 are undoubtedly close to the ventricle, since the P-waves resemble P_1 and P_2 . The form of QRS is abnormal in each case. The Q-wave is prominent in each, and there is no significant R-wave except at 60 cm. The RS-T segments are elevated, and the T-waves diphasic (plus-minus). At the last level (60 cm.) the QRS is of low voltage and is chiefly RS followed by an upright T-wave. As this probably is in the stomach and beneath the heart, it appears to reflect a more normal myocardium in that region.

In summary, the finding of deep Q in the E.V. lead, as well as the elevated RS-T segment and T-wave change, is sufficient evidence to base a diagnosis of acute myocardial infarction in this case.

Subacute Stage of Posterolateral Myocardial Infarction

R. B., a male actor, aged 64, was studied previously (Fig. 6a) on October 7, 1941. He developed bronchopneumonia requiring oxygen and sulfathiazol therapy for about one week. He showed progressive cardiac improvement. He had no complaints on the nineteenth day of his disease, when the following study (Fig. 6b) was made.

The standard electrocardiograms ($\frac{2N}{1}$ sensitivity) show persistent low voltage and deep Q_2 and Q_3 , a disappearance of elevated RS- T_2 and RS- T_3 segments, and a moderate inversion of T_2 and T_3 by comparison with previous study.

The unipolar extremity potentials ($\frac{2N}{1}$ sensitivity) show changes in each lead; however, the persistent Q, the disappearance of elevated RS-T, and appearance of inverted T in the LL lead are the most important. These suggest indirectly the presence of a posterior myocardial infarct.

The precordial potentials ($\frac{N}{T}$ sensitivity) show an abnormal, exaggerated R-wave at point 2 and possibly at point 3; however, the intrinsic interval is not definitely delayed at points 1, 2, or 3 over the right precordium. This rules out a change due to hypertrophy of the right ventricle. The QRS complex is bizarre and abnormal at point 6 on comparison of both tracings. The RS-T segments are now nearly iso-electric at points 1, 2, 3, 4, and 5, where they were depressed on October 7, 1941, and at point 6 the RS-T is also iso-electric; however, it was elevated previously (Fig. 6a).

The esophageal potential ($\frac{N}{T}$ sensitivity) at point 30 is considered above the auricle and not abnormal. Points 35, 40, and 42.5 are considered proximal to the auricle and essentially normal. Points 47.5 and 52.5 are considered opposite the ventricle posteriorly and proximal to the central area of infarction, if we may judge by the deep Q and inverted T in this region. Point 55.0 may be at the margin of the infarct, whereas points 57.5 and 60.0 are proximal to healthy heart muscle near the stomach or esophagus.

In summary, the comparison study of Fig. 6a and 6b show progressive changes in the standard and all the exploratory potentials within two and nineteen days after the onset of the infarction. The E. V. leads localized the posterior area of infarction, whereas point 6 on the precordium was indicative of incomplete lateral wall infarction; therefore, this is a combined lesion of posterior and lateral wall as judged by the exploratory electrocardiograms.

Subacute Stage of Posterolateral Myocardial Infarction

M. N., a man aged 62, was admitted to the hospital on September 30, 1941. He complained of an acute attack of abdominal soreness, weakness, perspiration, and dyspnea six days before entry. Dyspnea and weakness persisted. During the past year he has complained of frequent gas and sour eructations and precordial distress with exertion and meals. He has been unable to



SUBACUTE POSTEROLATERAL INFARCT

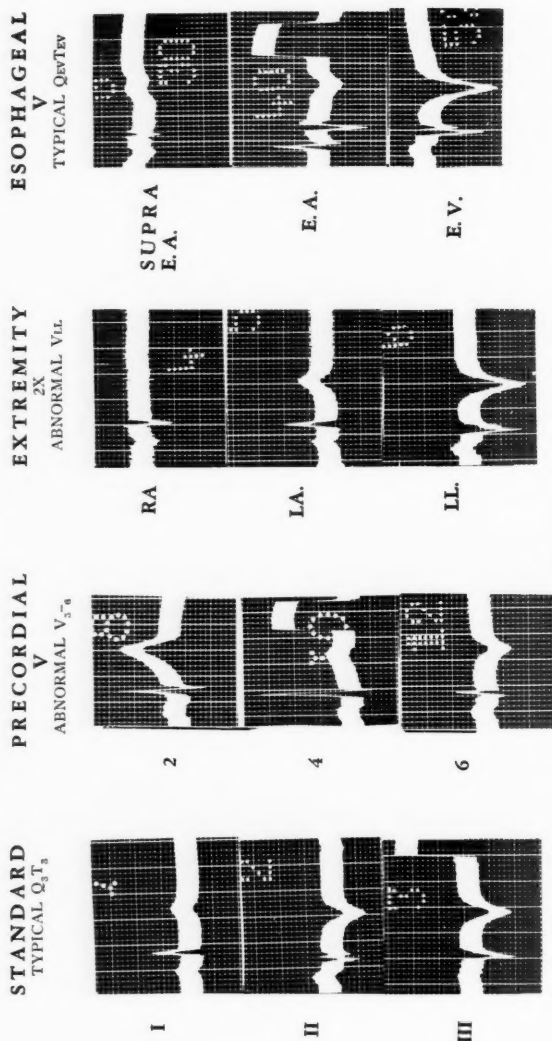


Fig. 7

Changes in this case of postero-lateral infarction are easy to follow. Note $Q_2 T_2$, $Q_3 T_3$, broad Q in III, LL, and EV, small Q and inverted T in V_6 . Whether initial R in LA is large and compensatory in origin or related to the horizontal electrical axis of the heart is hard to say. The abnormal Q patterns of infarction are usually irreversible and may be identified decades later.

work for the past year. Two weeks before entry a physician said his blood pressure was normal and stated that his symptoms were related to his heavy smoking. His blood pressure was "12 points below normal four year ago." During the first and second hospital weeks his sedimentation rate was elevated; however, he had a normal leukocyte count.

The standard lead electrocardiograms showed progressive changes of the Q_3T_3 type prior to the present study. These potentials ($\frac{N}{I}$ sensitivity) on October 17, 1941, (Fig. 7) show a typical Q_3T_3 pattern with a deep Q_2 and Q_3 , iso-electric RS-T segments, and sharply inverted T_2 and T_3 .

The single extremity potentials ($\frac{2N}{I}$ sensitivity) show marked variations from the normal in lead LL, in which Q is deep and the only QRS complex followed by a deeply inverted T-wave. This is indirectly suggestive of posterior myocardial infarction.

The precordial potentials ($\frac{N}{I}$ sensitivity) are essentially normal at points 1, 2, 3, and 4; however, R at point 2 appears taller than normal. There is no evidence of delay in the intrinsic interval over the entire precordium. QRS is not abnormal at points 5 and 6; however, the T-waves are abnormally inverted and suggest myocardial changes of the apical or lateral wall.

The esophageal potentials ($\frac{N}{I}$ sensitivity) are within normal limits at points 32.5, 37.5, 40.0, and 42.5, which are proximal to the auricle. At points 47.5, 50.0, 55.0, and 60.0, which are proximal to the ventricle, the entire QRS-T complexes are abnormal and indicate a large central area of infarction of the ventricle proximal to the esophagus and stomach wall.

In summary, the standard, unipolar extremity, precordial, and esophageal potentials are indicative of posterior and lateral myocardial infarction.

Incomplete Infarction of the Posterior Wall—Chronic

S. O., a man aged 62, (on March 7, 1938) suddenly began to have a burning pain in his chest. At home, he was relieved by a

INCOMPLETE POSTERIOR MYOCARDIAL INFARCTION

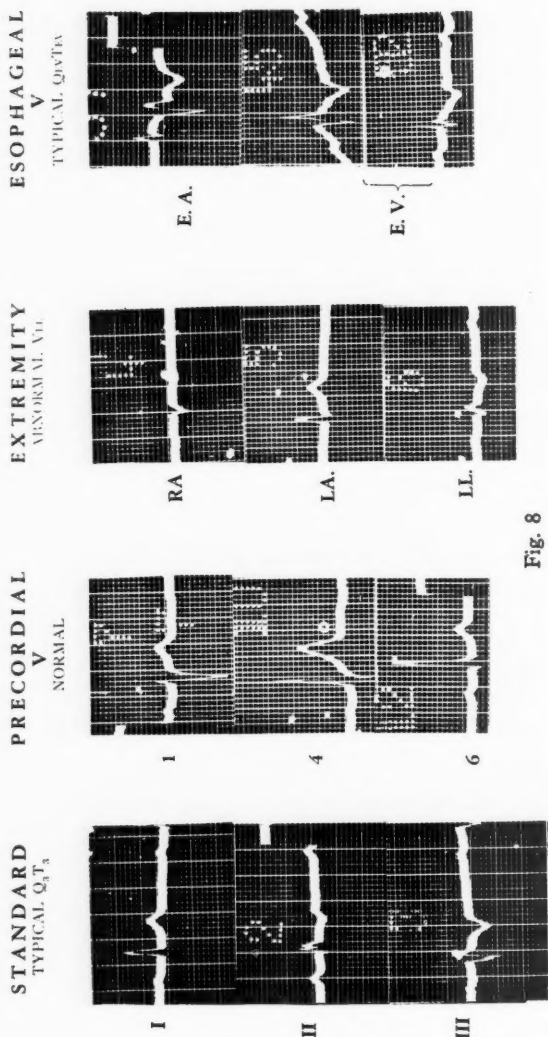


Fig. 8

Although the standard leads are diagnostic of posterior myocardial infarction, additional assistance is obtained from lead LL in establishing this diagnosis. The precordial leads are normal. The EV potentials suggest that the infarction involves only a portion of the posterior wall of the heart.

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"hypodermic" injection and bed rest. After nine days, however, he went out walking again and experienced a similar pain. A few weeks later he was studied in the cardiac clinic. His blood pressure was 162/94, the pulse rate was 84, the apex was 12 cm. from the mid-sternal line in the fifth intercostal space, a systolic murmur was constantly present at the apical region, by fluoroscopy the left ventricle was moderately enlarged and the aorta was markedly widened, and an electrocardiogram on April 6, 1938, showed first-stage A-V block with a PR interval of 0.22 seconds and a QRS of 0.08 seconds. A Q_3T_3 type of abnormality was indicative of a recent myocardial infarction.

During the following year marked clinical improvement was noted, while there was only occasional pain over the precordium on exertion, easily relieved by rest. His blood pressure was 120/90 on March 27, 1939. During November and December, 1940, he was hospitalized for furunculosis and diabetes, which was controlled without insulin. On September 26, 1941, he had no significant cardiac complaints. He walked vigorously. His blood pressure was 130/78 in the right arm and 126/82 in the left arm, the brachial arteries were tortuous, the heart tones were distant, while the systolic murmur persisted at the apex, and there were no signs of decompensation.

In September, 1941, (Fig. 8) a standard and complete exploratory electrocardiogram showed the following abnormalities: *the standard leads* ($\frac{N}{I}$ sensitivity) show the typical Q_3T_3 pattern with persistent small Q_2 , deep Q_3 , inverted T_2 and T_3 with a tendency to low QRS_{1, 2, and 3}.

The extremity potentials ($\frac{N}{I}$ sensitivity) show a prominent R in lead LA, while QRS in LL is bizarre, initiated by a small Q of 0.1 millivolts and followed by an inverted T-wave, suggesting an abnormality of the posterior myocardium.

The precordial potentials ($\frac{N}{I}$ sensitivity) are normal from all conventional points (V_1 - V_6).

The esophageal electrocardiograms are normal from points proximal to the auricle (Fig. 8: 32.5, 35.0, 37.5, 40.0, and 42.5); however, opposite the ventricle (45.0, 47.5) the curves are abnormal in that the Q-wave is approximately 0.6 millivolts or greater than 50% of the R deflection in the same lead. At lower levels (52.5), possibly in the stomach, the QRS appears normal, but the T-wave is inverted and abnormal. These changes in the posterior ventricular region resemble those curves obtained from the border-line of infarcts and areas of incomplete infarction of the myocardium shown by Wilson, et al, (21) in animals.

In summary, this case shows an area of myocardial infarction posteriorly by the esophageal lead about two and one-half years after the onset of the attack. Whenever lead LL patterns are obtained similar to this case, the author feels that one is dealing with a pathological case, probably that of infarction. In this case the standard leads serve as sufficient warning that grave pathology is present.

*Atypical Posterior Myocardial Infarction—Chronic
(Equivocal Standard Leads in 1941)*

R. R., a man aged 42, a heavy cigarette smoker, experienced pain across the chest with radiation to both arms, for a period of ten minutes, for the first time 1½ months before study in the cardiac clinic (October 3, 1938). Following this episode, the pain was produced only while taking a hot bath, but not by exercise until much later. His blood pressure was 138/90, pulse rate was 78 and regular, and there was no evidence of enlargement of the heart, although the cardiac-thoracic ratio was 50%. Gradual improvement took place. The patient has taken frequent airplane flights without any untoward cardiac symptoms. In 1941 there was an occasional tight feeling across the precordium on vigorous exercise. His blood pressure was 130/76, and the heart on recent fluoroscopy was normal in contour, while the cardiac-thoracic ratio remained 50%. This is a case of coronary sclerosis with healed coronary thrombosis. There is also moderate peripheral sclerosis of the radial and brachial arteries.

ATYPICAL POSTERIOR MYOCARDIAL INFARCTION
EQUIVOCAL STANDARD LEAD

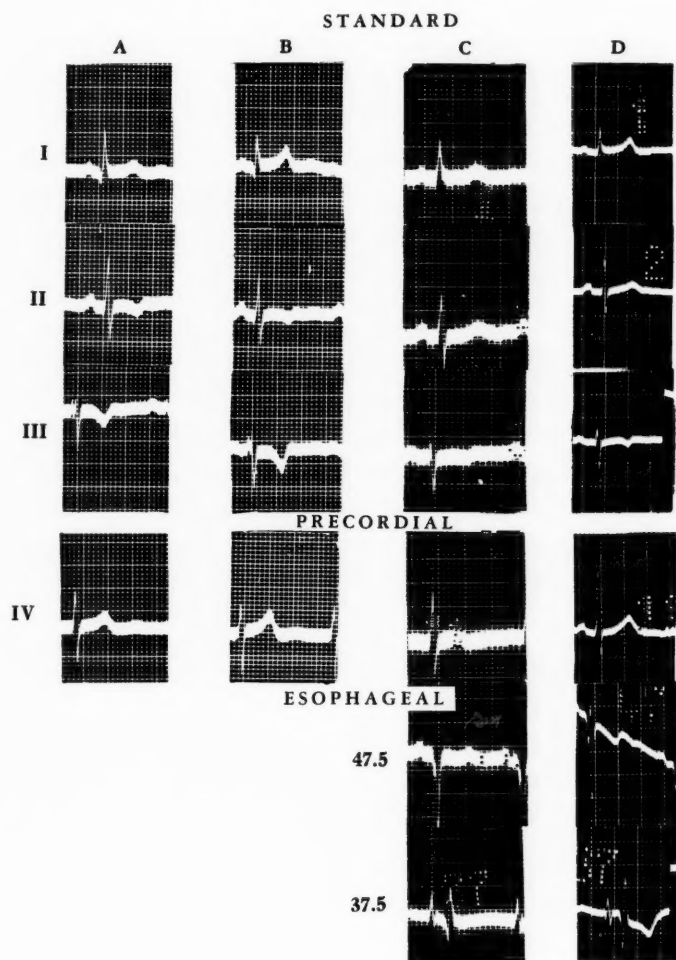


Fig. 9A

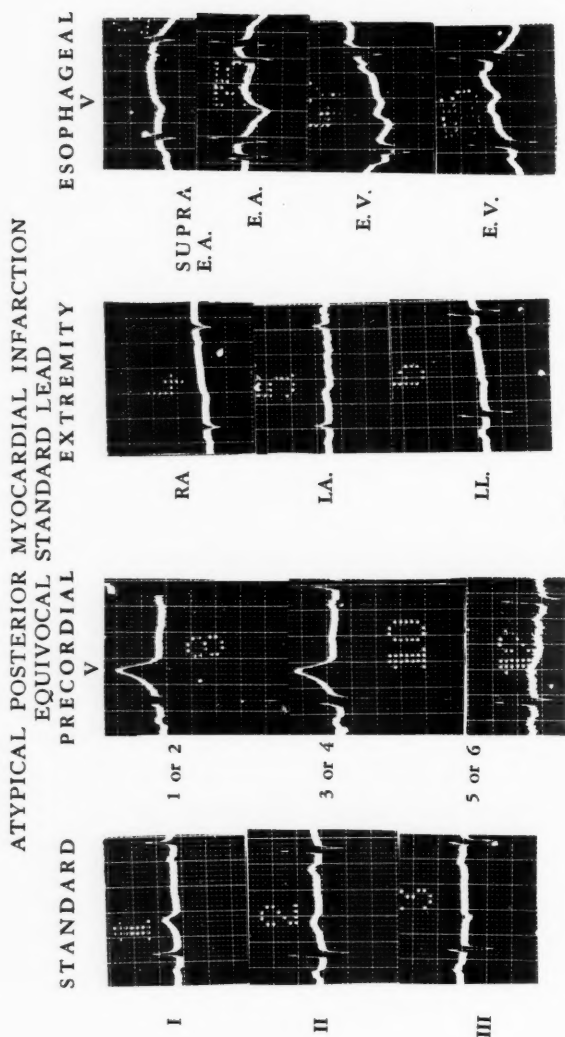


Fig. 9B

The T_a patterns in posterior myocardial infarction are reversible if we may judge by the standard leads of this case. The transitions from A to B to C to D confirm this. Standard leads in D are considered normal; its lead IV is normal but a pathological change is revealed by esophageal ventricular leads at 47.5 cm. from the nares in C and D. Thus the equivocal standard leads of this type keep us ignorant of the true pathology.

The infarct involves a small portion of the posterior wall as judged from the esophageal studies.

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A preliminary group of electrocardiograms (Fig. 9a) shows the progress of this case by standard and selected exploratory leads over the course of three years. A T_3 type of tracing is present with a normal Lead IV on October 7, 1938 (A), and on November 11, 1938 (B). In both of these T_2 is also inverted. A deep S_2 and S_3 suggest left axis deviation. By June 2, 1940 (C), the inverted T_2 has disappeared; however, a slightly inverted T_3 persists. Therefore, the electrocardiogram has become equivocal for any evidence of myocardial pathology by the standard and precordial leads. These leads remain equivocal in 1941 (D).

An esophageal study (Fig. 9a) in 1940 (C) shows an abnormal Q as the only QRS deflection in the E. V. lead at 47.5 cm. from the nares. This is followed by a low, upright T-wave. The E. A. lead shows a typical P-wave and a normal QRS; however, an iso-electric T-wave at 37.5 cm. is present. In 1941 (D) the selected E. V. leads show changes since previous study. It is noted that QRS in the E. V. lead is notched and is followed by an inverted T, both of which are abnormal. Lead E. A. is normal at this time. The next illustration will show multiple exploratory leads on this same case.

Atypical Posterior Myocardial Infarction—Chronic

The history and findings on this case are discussed with the preceding illustration (Fig. 9a.). The following should be noted in Fig. 9b:

The standard leads are equivocal for myocardial infarction; however, they show a left electrical axis deviation and probably a normally inverted T_3 .

The unipolar extremity potentials ($\frac{N}{I}$ sensitivity) are normal in RA and LA; however, LL shows an abnormally diphasic or inverted T-wave. This is presumptive evidence of some myocardial involvement, since in the normal table the T-wave in lead LL is always electropositive.

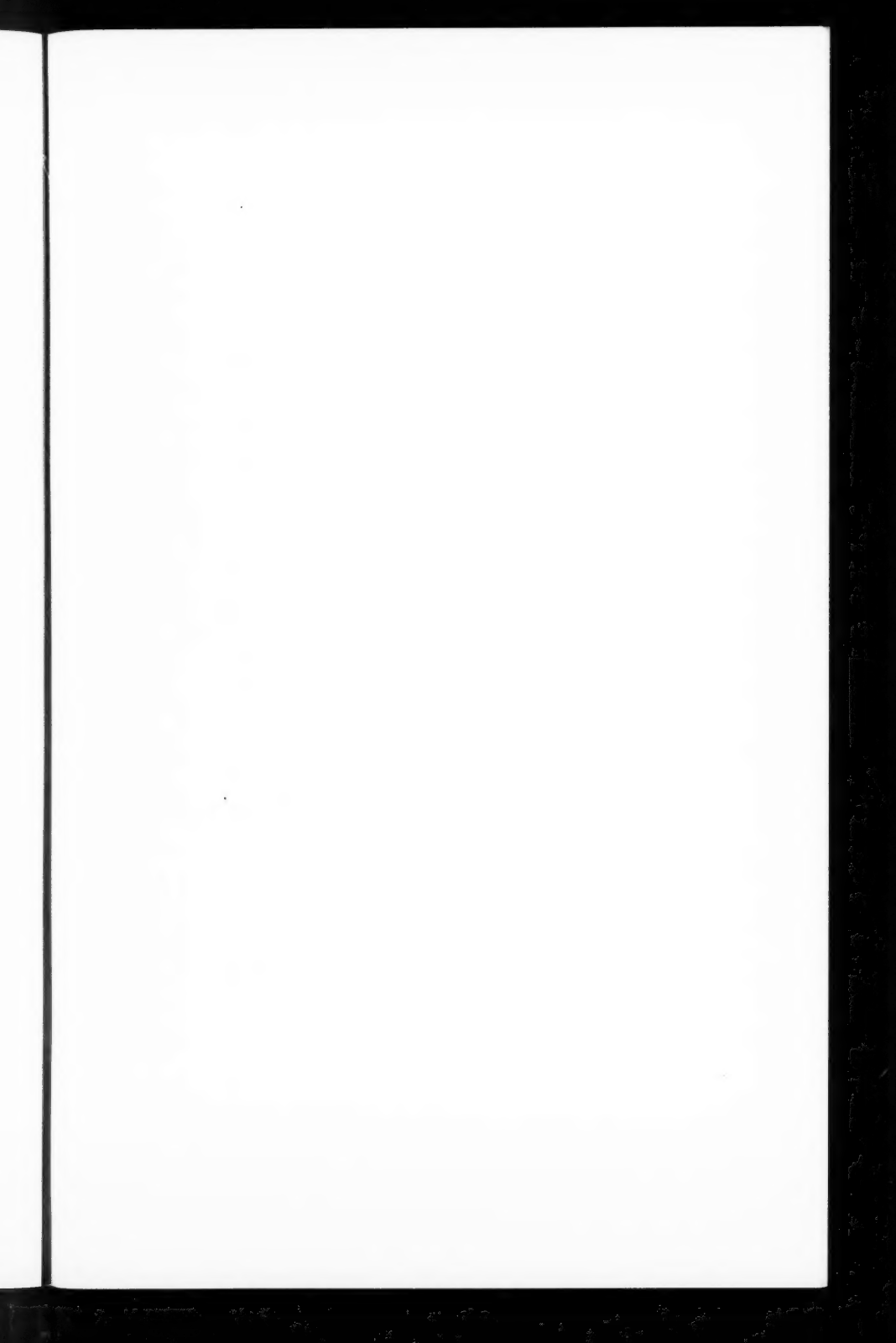
The precordial series of potentials ($\frac{N}{I}$ sensitivity) are normal from points 1, 2, 3, 4, and 5. The T-wave at point 6 appears to be only 0.1 millivolts electropositive; this is near the lower limits of normal in this position.

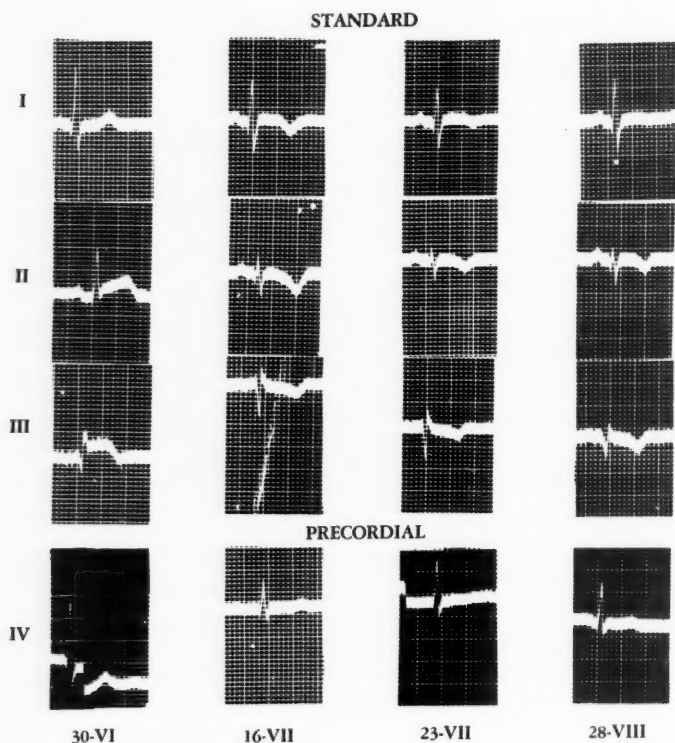
The esophageal leads ($\frac{N}{I}$ sensitivity) are normal from point 30 above the auricle and essentially normal from points 32.5, 37.5, 40.0, and 42.5, which are proximal to the auricle. In this region, however, the Q-wave is usually larger than R, rather than smaller, as is noted here. At lower points 45.0, and 47.5, if we may judge by the form of the P-wave, we are opposite the ventricle in the esophagus and stomach. At points 45.0 and 47.5 a Q-wave of 0.5 millivolts is followed by a small R of about 0.2 or 0.3 millivolts, a deep S of 1.5 millivolts, and a definitely inverted T-wave. These abnormalities are indicative of localized myocardial changes or infarction in this region. At lower levels (55.0) QRS and T-waves are essentially normal, and, therefore, no evidence of localized pathology is present at this region.

In summary, myocardial pathology is localized by the E. V. and LL leads, although the standard and precordial leads are equivocal. Only the upper portion of the esophageal ventricular region appears to be abnormal. Such localized infarctions are more frequently encountered on the anterior wall. In insurance underwriting the standard and precordial leads would be accepted as normal.

Posterolateral Myocardial Infarction Mixed QT Pattern

G. B., a man aged 42, was admitted to the hospital on June 30, 1941, because of severe substernal pain without radiation, lasting for twelve hours before partial relief was obtained from two "hypodermic" injections. A similar and first attack of pain lasting two hours occurred two weeks previously. This was relieved by one "hypodermic." A relevant past history of hypertension, given as 140/110, and post-scarlatina albuminuria was present since 1925. After hospitalization of eleven weeks and discharge,





POSTERIORLATERAL INFARCT

MIXED QT PATTERN

Fig. 10A

Exploratory Electrocardiograms 75

he felt well; however, he was conscious of occasional extrasystoles. His blood pressure was 105/80, his pulse rate 100, and there was a loss of 25 pounds in weight. His heart tones were normal, while the fluoroscopy of his heart showed no evidence of enlargement except generally of the aorta. A paradoxical pulsation of the left ventricle was noted in the left oblique view by one observer under the fluoroscope.

A series of standard ($\frac{N}{1}$ sensitivity) and single precordial lead electrocardiograms ($\frac{N}{2}$ sensitivity) demonstrate clearly (Fig. 10a) the progressive changes seen in myocardial infarction. On June 30, 1941, they show a slight lowering of RS-T₁, an elevated RS-T₂ and RS-T₃, a (plus-minus) diphasic T₃ as well as a small Q₃ of 0.2 millivolts, which is normal, and lastly, an abnormally depressed RS-T₄ of 0.4 millivolts. These findings are suggestive of acute involvement of the posterior myocardium.

On July 16 there is no longer an RS-T displacement; however, T₁, T₂, and T₃ have become inverted, while T₄ has become nearly iso-electric. QRS₂ has become small and W-shaped, and QRS₃ is definitely M-shaped.

On July 23 there is no further change in QRS_{1, 2, 3}, and ₄; however, T₁, T₂, and T₃ have regressed toward the iso-electric line but remain inverted.

On August 28 QRS₃ has become slurred, while T₁ is now iso-electric, and T₂ and T₃ remain inverted. There is no conclusive evidence of a deep Q in any lead. Lead IV is of the normal type with a tendency to a low T₄.

In summary, it is clearly seen that the changes in the standard and precordial leads are not typical of an anterior or a posterior infarct. This atypical case of infarction is analyzed by multiple exploratory leads in the succeeding graph (Fig. 10b).

Posterolateral Myocardial Infarction Mixed QT Pattern

The major changes in case G. B. which took place in the standard leads are illustrated in the previous figure (10a), beginning

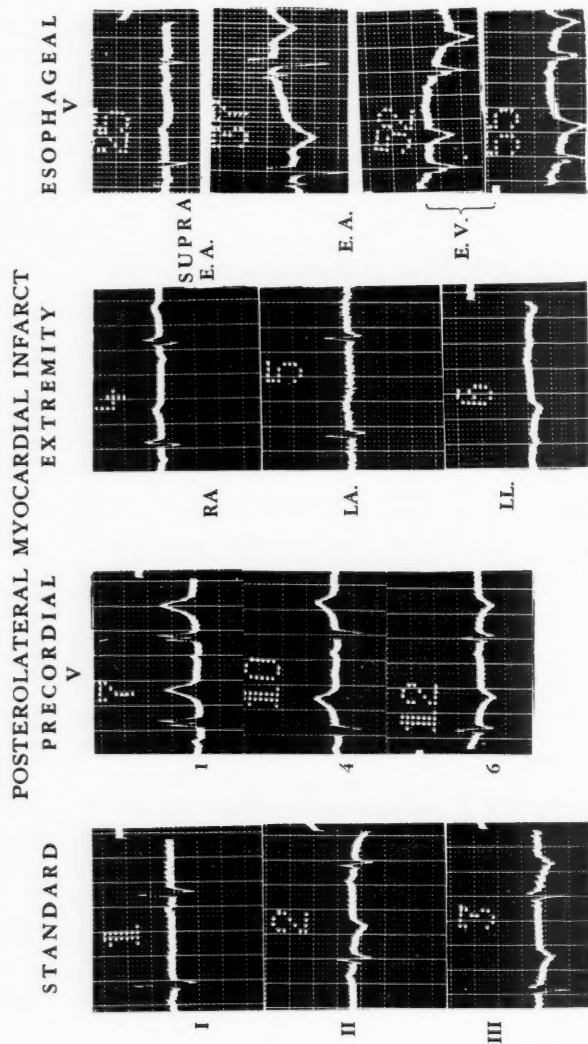


Fig. 10B

The progressive changes in the standard leads are difficult to evaluate without the addition of multiple exploratory leads. The changes in lead I are due to changes in the lateral wall. The changes in II and III and LL are due to complete infarction of the posterior ventricular region.

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two weeks after the onset of the acute coronary disease and continuing through the twelfth week. During the following week (September 6, 1941) the inclusive investigation was made (Fig. 10b).

The standard leads ($\frac{N}{I}$ sensitivity) show a normal QRS complex, RS-T segment, and an abnormal, iso-electric T-wave in Lead I. A persistent W-shaped QRS and an inverted T are present in Lead II. Lead III is initiated by a small R-wave, followed by an S-wave of 0.5 millivolts and a second R-wave, giving the complex an M-shaped appearance. T_3 is also inverted. These findings do not localize the myocardial infarct.

The single extremity potentials ($\frac{N}{I}$ sensitivity) show an abnormal, upright T in lead RA, an abnormal Q in lead LL (which is the only QRS complex in this lead), followed by an abnormally inverted T. These findings suggest a partial localization of an infarct on the posterior myocardium.

The precordial potentials ($\frac{N}{I}$ sensitivity) are of interest over point 1 because of the exaggerated, notched R-wave in the absence of S, followed by a T-wave of 0.7 millivolts, which is maximum for this lead. Points 2, 3, and 4 are within normal limits of Table III. At points 5 and 6 the QRS complexes are normal; however, they are followed abnormally by sharply inverted T-waves. These latter findings are sometimes found over areas of incomplete infarction or at the border-line of an infarct; therefore, it may be concluded that the lateral or apical region is definitely altered by some abnormal factor.

The form of the esophageal potentials ($\frac{N}{I}$ sensitivity) at point 25 resembles lead RA in all respects. At points 30 and 32.5 an artefact is superimposed on the T-wave with each mechanical systole. Points 32.5, 37.5, and 40.0 are normal indirect leads from the auricle. Point 42.5 is considered border-line between the auricle and ventricle. Points 45.0, 47.5, 50.0, 52.5, and 55.0 are considered to be indirect leads from the posterior and diaphragmatic

ANTERIOR AND POSTERIOR INFARCTION

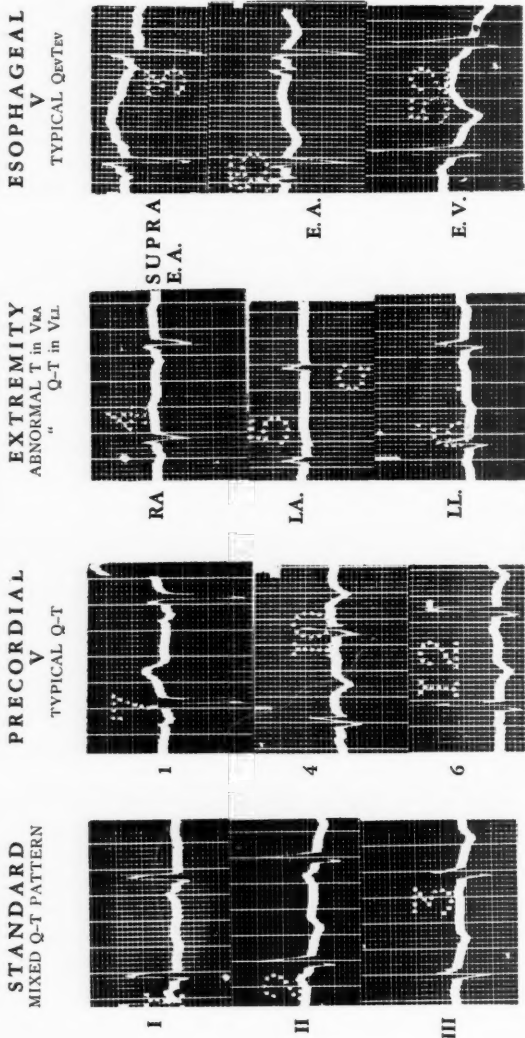


Fig. 11

The findings in this case suggest incomplete myocardial infarction of the anterior, lateral, and posterior walls. Lead LL is helpful in confirming this. The initial R spike and upright T wave in lead RA are often seen in the presence of anterior myocardial infarction.

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region of the ventricle. These are all abnormal. Q is the major and only QRS complex, and it is followed by a deeply inverted T-wave at each point. These last findings demonstrate a significant lesion in the posterior ventricular region.

In summary, in the light of findings by exploratory leads we may be certain that the changes in the standard leads were wholly significant. They were indirectly diagnostic of a posterolateral myocardial infarct as demonstrated specifically by the esophageal ventricular and (V_5 - V_6) precordial leads on the lateral wall.

Myocardial Infarction of the Anterior and Posterior Walls

K. L., a night watchman at The Connecticut Mutual, aged 50, suffered severe vise-like cramps across his chest on Thanksgiving night (November 27, 1940) just as he was going on duty. These pains continued for one and one-half hours when he was relieved by two "hypodermic" injections. He was hospitalized immediately, and a standard-lead electrocardiogram was diagnostic of myocardial infarction. His progress was satisfactory for one week, when he suffered a second severe attack of precordial distress. This was relieved by one "hypodermic" injection and "sleeping tablets." He was discharged as well after six weeks and has since returned to his usual work without symptoms. On recent fluoroscopy, a moderate left ventricular enlargement was noted; paradoxical pulsations were not made out. The present electrocardiogram (Fig. 11) was taken on September 10, 1941, which is about ten months after the onset of his symptoms.

The standard potentials ($\frac{N}{T}$ sensitivity) are atypical for anterior or posterior myocardial infarction but are of interest when analyzed with the exploratory leads. Lead I shows an abnormal iso-electric T-wave. Lead II shows a deep Q-wave followed by a tall R and inverted T-wave. Lead III shows similar changes to Lead II. This cannot be classified as a typical Q_2T_3 type of curve on account of the atypical Lead I.

The single extremity potentials ($\frac{N}{T}$ sensitivity) vary from the normals in RA and LL. An abnormal, upright T-wave is present in RA. QRS in LL has an initial deep Q of 0.25 millivolts, which

is outside normal limits (of Table I). An abnormally inverted T-wave of 0.1 millivolts is also present in LL. The LL findings strongly suggest pathology on the posterior wall, where lead RA suggests possible anterior myocardial changes. Initial R in RA is more positive than Q in LA; therefore, Q_1 is present.

The precordial potentials ($\frac{N}{T}$ sensitivity) are normal at point

1. At point 2 the R-wave is smaller than at point 1 and is, therefore, abnormal. At point 3, the deep Q-wave has replaced the normal QRS complex and suggests a "central area" of myocardial infarction. At points 4, 5, and 6 the Q-wave is about 0.4 millivolts and large in proportion to each respective R-wave. The T-waves are also abnormal and inverted at points 4, 5, and 6. These types of curves are often obtained at the margin of infarcts, as observed previously.

The esophageal potentials ($\frac{N}{T}$ sensitivity) at point 30 resemble RA and are abnormal with respect to T in regions above the auricle. Point 35 has a normal QRS and T deflection. Points 40 and 45 may also be considered proximal to the auricle and are abnormal with respect to exaggerated R-waves and upright T-waves. Points 47.5, 50.0, and 55.0 represent abnormal potentials from the region of the ventricle, since there are prominent Q-waves present of about 0.5 millivolts or greater, as well as inverted T-waves at these levels. These curves are similar to the precordial potentials from points 4, 5, and 6 and suggest areas of incomplete infarction beneath the electrodes in the posterior ventricular region.

In summary, there is evidence of complete myocardial infarction of the anterior wall and incomplete infarction of the lateral or apical, and posterior myocardium by the exploratory leads. This explains the atypical standard potentials.

Atypical Myocardial Infarction—Anterior and Posterior

M. R., a man aged 61, was first observed in the Post-Graduate vascular clinic in 1940, where he was treated for varicose veins. A hypertension was first noted at this time, and a routine elec-

ANTERIOR AND POSTERIOR MYOCARDIAL INFARCTION

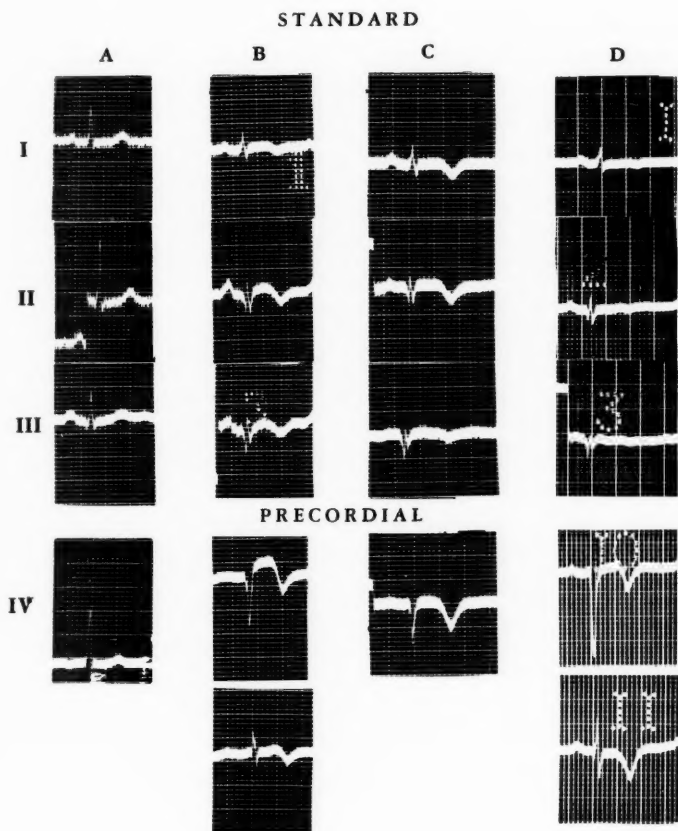


Fig. 12A

Progressive changes in the standard leads of this case are difficult to interpret without the assistance of the exploratory leads.

ANTERIOR AND POSTERIOR INFARCTION

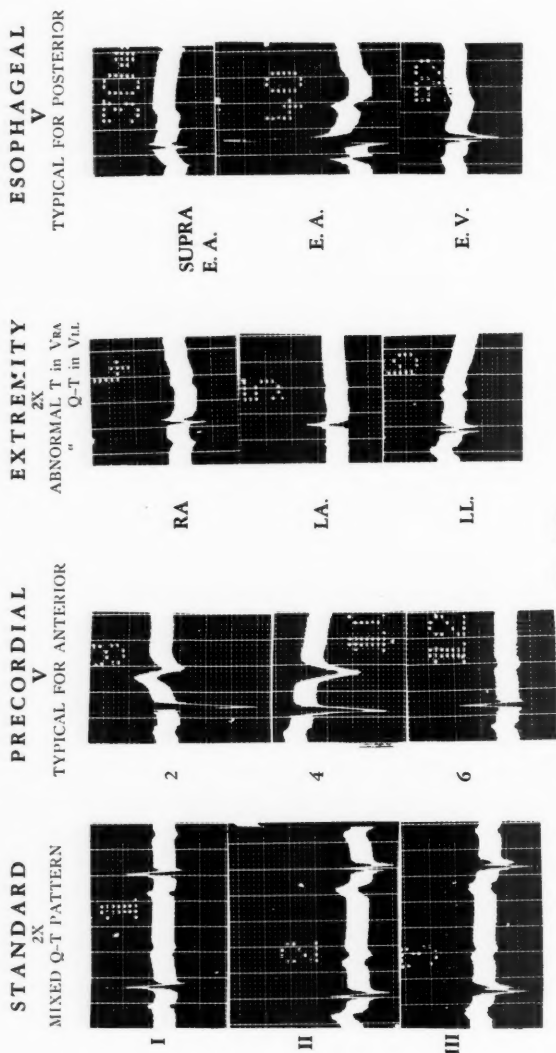


Fig. 12B

More than one infarct often exists and produces mixed and peculiar Q-T patterns in the standard leads. In this case, we have changes in I, IV, and RA, reflecting an anterior lesion, and changes in II, III, LL, and EV, reflecting a posterior lesion. R is abnormal and usually tall in the EA lead.

trocardiogram (Fig. 12a) on November 23, 1940, (A) showed no evidence of myocardial changes. There was a normal electrical axis of about 60 degrees, and the T-waves were upright in the conventional four leads. It was interpreted within normal limits.

On entry (May 10, 1941) (B) to the hospital because of persistent weakness, he complained of having had a severe heart attack occurring at work about two weeks previously. A squeezing pain, across the upper chest and radiating to the back only, had lasted for about 20 to 30 minutes. On admission his blood pressure was 168/98, his rhythm was regular, and his heart was enlarged to the left. On May 14, 1941, his blood pressure was 118/70, and the sedimentation rate was elevated to 48 mm. per hour (Wintrobe). As his clinical progress was satisfactory, he was discharged after six weeks and then followed in the cardiac clinic. Except for rales at the left base and moderate left ventricular enlargement by fluoroscopy, there was no evidence of clinical decompensation on September 27, 1941. Digitalis was not prescribed during this study, which is reported in two figures (12a, 12b).

The standard leads ($\frac{N}{I}$ sensitivity) two weeks after (May 10, 1941) (B) his attack of pain, show a marked reduction of voltage in Leads I, II, and III (Fig. 12a), a W-shaped QRS_2 and QRS_3 , a slightly elevated $RS-T_2$ and $RS-T_3$, and inverted T_1 , T_2 and T_3 . *Precordial leads* ($\frac{N}{2}$ sensitivity) at V_4 show a deep Q-wave, an elevated RS-T segment, and a (plus-minus) diphasic T-wave. At V_5 a low voltage QRS complex, an iso-electric RS-T segment, and an inverted T-wave are present. Progressive changes in the standard and precordial leads are compared in this same figure on May 23, 1941 (C), and on November 27, 1941 (D). Although Lead IV in this case localizes an anterior myocardial infarct, Leads II and III are suggestive of a posterior infarct; therefore, this subject is investigated with detailed exploratory leads. These justify the other findings in the standard leads (Fig. 12b and intermediate discussion).

Atypical Myocardial Infarction — Anterior and Posterior
(No Figure)

The standard leads ($\frac{N}{I}$ sensitivity) of M. R., aged 61, on May 17, 1941, show a conglomeration of changes. Lead I is consistent with the T_1 data originally described by Parkinson and Bedford, whereas Leads II and III are consistent with the Q_3T_3 data described by Wilson.

The precordial potentials ($\frac{N}{I}$ sensitivity) at this time support the diagnosis of anterior myocardial infarction, since there are deep Q-waves, slightly elevated RS-T segments, and sharply inverted T-waves at points 2, 3, 4, and 5. Point 6 shows a deeply inverted T-wave as the chief abnormality, and this is probably not over the central area of infarction, like the preceding potentials.

The esophageal potentials ($\frac{N}{I}$ sensitivity) using the left leg as the indifferent electrode, are abnormal above and opposite the auricle at points 25, 27.5, 30.0 32.5, and 37.5, but are difficult to evaluate. Opposite the ventricular region, as judged by the character of the P-wave, the potentials from points 42.5, 45.0 47.5, 52.5 and 57.5 are also markedly abnormal. In each of these potentials a deep Q-wave, which is the only QRS complex, is followed by an inverted T-wave and resembles the form of those potentials on the precordium at points 2, 3, 4, and 5.

In conclusion, it appears that there are two large central areas of infarction, one on the anterior wall and the other on the posterior wall. These observations are confirmed by the next electrocardiographic study (Fig. 12b).

Atypical Myocardial Infarction — Anterior and Posterior

A second detailed study by standard and exploratory electrocardiograms of M. R., aged 61, was made on October 7, 1941. This was about five and one-half months after the acute onset of coronary symptoms in this case (Fig. 12b).

The standard potentials ($\frac{2N}{I}$ sensitivity) show persistent low voltage and bizarre QRS complexes; however, T_1 is nearly isoelectric, T_2 is diphasic, and T_3 is now upright by comparison with previous studies (Fig. 12a). These findings, apparently favorable to the patient and consistent with clinical improvement, are difficult to evaluate without exploratory leads.

The single extremity potentials ($\frac{2N}{I}$ sensitivity) show an abnormal, upright T-wave in RA suggestive of possible changes on the anterior wall. Lead LA is normal; however, LL shows an abnormal, W-shaped Q-wave followed by a diphasic T-wave. This suggests a probable infarction of the posterior myocardium.

The precordial potentials ($\frac{N}{I}$ sensitivity) are essentially normal at points 1 and 6; however, points 2, 3, 4, and 5 are persistently abnormal as compared with the intermediate study of this case. These are typical of anterior myocardial infarction.

The esophageal potentials ($\frac{N}{I}$ sensitivity) using the central terminal as the indifferent electrode, at points 30.0, 35.0, 40.0, and 45.0, which are above or opposite the auricle, are also persistently abnormal and difficult to evaluate; however, in view of the marked damage to the myocardium, it is reasonable to believe that the form of the intraventricular potential varies greatly. It is believed that the esophageal auricular leads are the best index of this change. At points 47.5, 50.0, and 52.5 the electrode is again placed proximal to the ventricle. These potentials are abnormal and indicative of a central area of infarction if we may judge by the deep and notched Q-waves followed by inverted T-waves in this region.

In summary, the several electrocardiograms of this case reveal evidence supporting a diagnosis of combined anterior and posterior myocardial infarction.

An Equivocal Case of Probable Posterior Myocardial Infarction

A. L., a woman aged 54, complained of an attack of dizziness and weakness with a feeling of pressure in the left chest on April

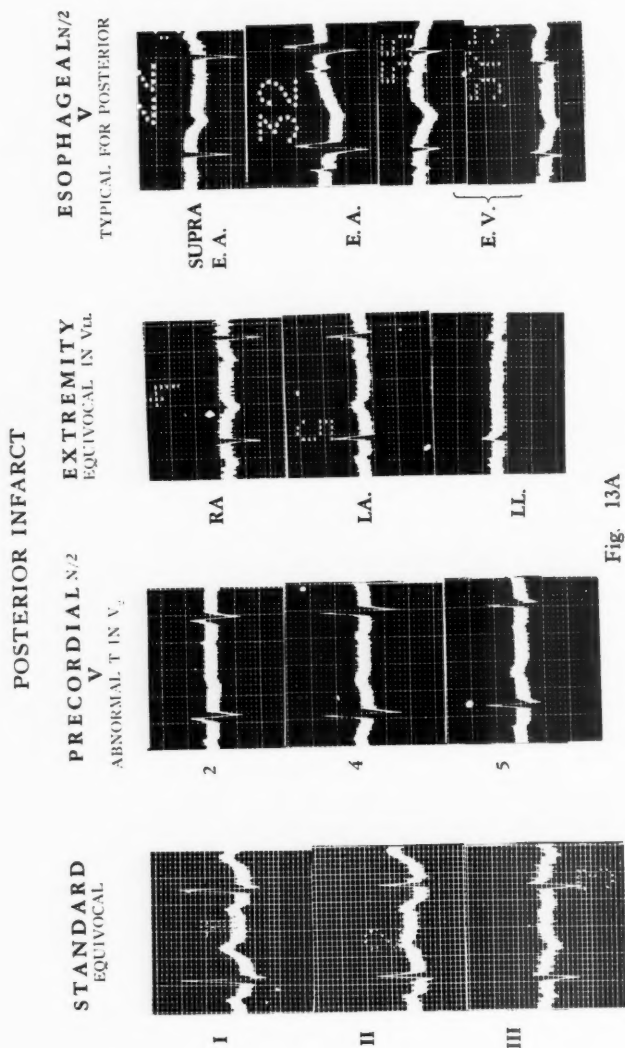


Fig. 13A

2, 1938. She was not confined to bed. She weighed 177 pounds and was considered overweight. Since April, 1938, she was able to do physically whatever she wished without any discomfort. On dietary measures she reduced her weight to 150 pounds by October, 1938; however, she regained gradually to 157 pounds by May, 1941. Her blood pressure has been within normal limits. During 1939, 1940, and 1941 she has been free of any precordial discomfort. Complete standard and exploratory electrocardiograms were obtained in April and in November, 1938. These will be discussed in view of the critical changes which took place, particularly in the esophageal leads opposite the ventricle.

The first study on April 5, 1938, was three days after the precordial attack. *The standard leads* ($\frac{N}{T}$ sensitivity) in Fig. 13a show a normal QRS_1 and QRS_2 ; however, QRS_3 is peculiar in that it is difficult to decipher whether it is initiated by Q or R. Slight left axis deviation is present. The RS-T segments and T-waves are within normal limits. The second study on November 10, 1938 (Fig. 13b), shows no conclusive change in the standard leads; however, QRS_3 appears to be composed of a Q-wave only. Q_2 is about 0.05 millivolts; however, T_2 is persistently upright. The standard leads are thus considered equivocal.

The unipolar extremity potentials ($\frac{N}{T}$ sensitivity) do not assist the interpretation much; however, in LL the QRS complex is bizarre on April 2 and November 10, while the T-wave becomes definitely upright in November (13b) instead of remaining iso-electric. Hence, there is a suggestion of myocardial change in this course of about seven months. The large R in LA produces the deep Q_3 .

The precordial potentials ($\frac{N}{2}$ sensitivity) show normal QRS complexes over points 1 to 6 on both examinations. The RS-T segments are nearly iso-electric on both occasions at these same points. The T-waves show minor changes by comparison with each other. At points 1 and 2, T remains inverted; however, at point 3 it changes from inverted to upright position in the latest electrocardiogram (Fig. 13b). The T-waves at points 4 to 6 are

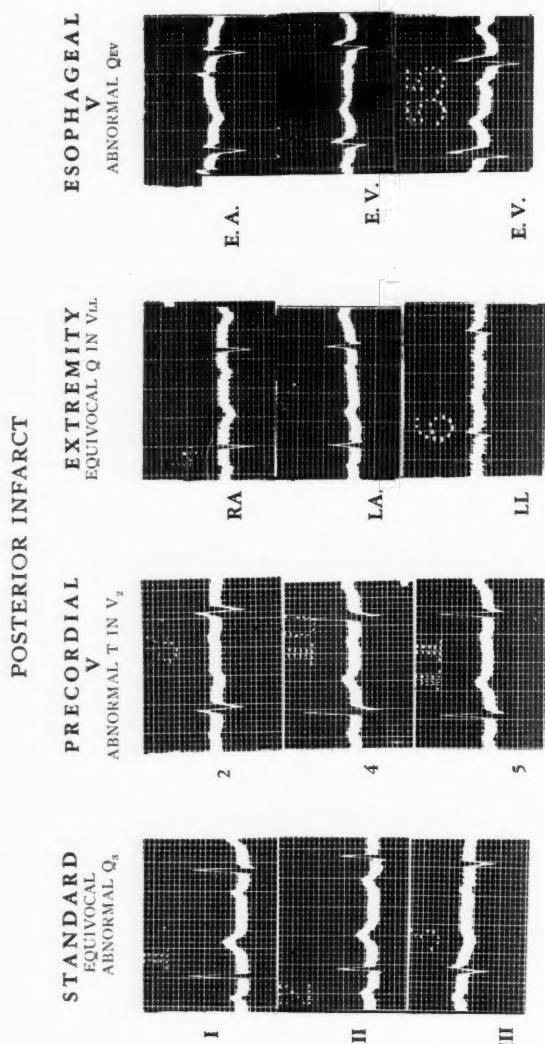


Fig. 13B

The decision of normality or abnormality of Q_3 patterns in stocky subjects is sometimes made by serial electrocardiograms. It may be also made by detailed exploratory leads at one investigation. In this case, Q_3 is associated with a peculiar qr in lead LL and a definitely abnormal esophageal ventricular potential, suggesting posterior infarction. Later study confirms the Q wave abnormality was persistent but the T wave abnormality transient in the EV leads.

more upright and electropositive on the second examination. These findings are inconclusive and are mainly in the region above the right ventricle. Recent studies by Wood (22) on pulmonary embolism describe similar non-specific changes at the left parasternal border, but they were described as reversible over a short period of time. In general, we must conclude that we have, thus far, no evidence of myocardial infarction.

The esophageal potentials ($\frac{N}{2}$ sensitivity) at points 20.0, 25.0, 30.0, and 35.0 show no significant variations from the normal for these levels. The auricular intrinsic deflection disappears at point 37.5, which may be considered as border-line between the auricle and ventricle. Below this level the P-waves are smooth and upright and, therefore, identify the juxta-ventricular leads. Thus, at points 40.0, 45.0, 50.0, 55.0, and 60.0 on April 5, 1938 (Fig. 13a), the Q-wave appears to be the most prominent deflection of the QRS complex, although it rarely exceeds 0.4 to 0.6 millivolts. At levels 50.0, 55.0, and 60.0 an abnormal, inverted T-wave is present. These changes seem to indicate an abnormal myocardium in this region. In the course of the second examination (Fig. 13b), persistent deep Q-waves are found at points 40.0 to 55.0; however, the T-waves at each point are now definitely electropositive and upright. Point 55.0 was taken at normal sensitivity and shows the typical configuration to better advantage. Thus, it appears that by virtue of the abnormal Q-wave in the E. V. leads at several levels and the changes in the T-waves of these leads over the course of several months' time we were dealing with a localized posterior myocardial infarction.

Right Ventricular Hypertrophy

F. D., a man about age 25, has congenital heart disease. Pulmonary stenosis is the major pathology, associated with marked right ventricular enlargement and shift of the apex to the left by fluoroscopy. Marked generalized cyanosis is present. No evidence of decompensation has been present. The standard electrocardiograms over several years have always shown marked right axis deviation.

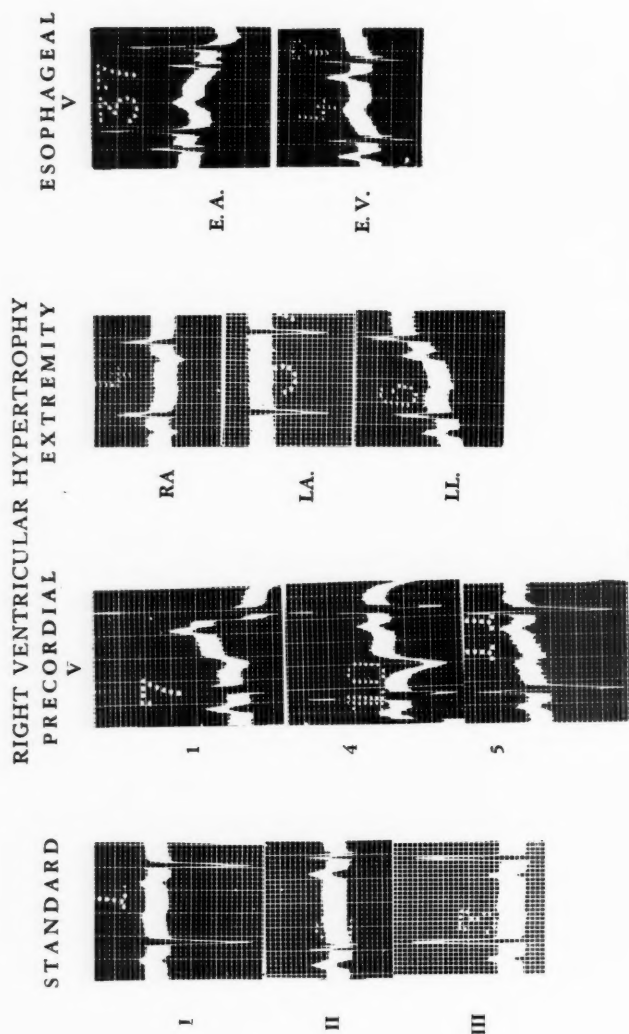


Fig. 14

Congenital pulmonary stenosis is probably responsible for the patterns of right axis deviation in this case. The large R wave on the right precordium is characteristic of right ventricular hypertrophy.

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The standard electrocardiograms ($\frac{N}{I}$ sensitivity) in the present study (Fig. 14) show tall and sharply spiked P_2 -waves of 0.3 millivolts. A right axis deviation of $+148^\circ$ with the horizontal exists. The PR interval is 0.14 seconds, and the QRS interval measures between 0.08 and 0.10 seconds. The T-waves are all of low voltage and less than 0.1 millivolts. T_1 is upright, T_2 is iso-electric, and T_3 is inverted. In right ventricular hypertrophy T_2 and T_3 are often simultaneously inverted.

The extremity potentials ($\frac{N}{I}$ sensitivity) show QRS changes consistent with marked right axis deviation and a heart probably rotated toward the spine, as judged by the tall R-spike in RA (12), (6), (25), and the deep S in LA. The T-waves are nearly iso-electric in each lead and, therefore, abnormal.

The precordial potentials ($\frac{N}{I}$ sensitivity) are characteristic of right ventricular hypertrophy. A small initial Q-wave is present at point 1 only. This is followed by a tall R-wave and relative delay in the intrinsic interval (0.05 seconds as compared with 0.02 seconds as the average normal). Exaggerated R is also present at points 2 and 3. Abnormal, sharply inverted T-waves are present at points 1, 2, 3, and 4, presumably proximal to the right ventricle of this case. Points 5 and 6 are more indicative of potentials near the left ventricle, judging by the onset of the intrinsic interval of about 0.03 to 0.04 seconds, which is normal for this region. The S-waves are unusually deep for this region; however, the T-waves are essentially upright and of low voltage (judging from a poor record). In summary, these findings are consistent with hypertrophy changes mainly involving the right ventricle as judged by the form of the abnormal right precordial potentials.

The esophageal potentials ($\frac{N}{I}$ sensitivity) are abnormal in type above (point 30.0) and opposite (points 32.5, 35.0, and 37.5) the auricle if we may judge by the chief deflection (R) and upright T-wave. Points 40.0 and 42.5 are presumably borderline in type, and in this case the QRS does not resemble those

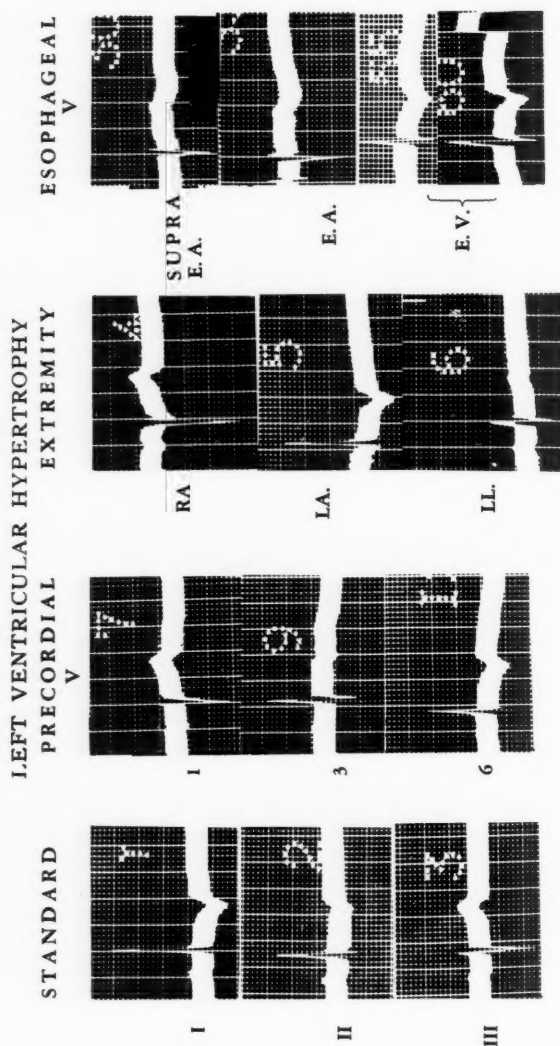


Fig. 15

Marked hypertension is probably responsible for the left axis deviation electrocardiogram. The T waves are inverted in the precordial and esophageal left ventricular potentials. Apparently, there is a generalized ischemia of the left ventricle without significant change in the QRS complex in these same regions.

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above or below these levels or any leads on the precordium; however, deep Q-waves are normally expected in the auricular region. The selection of points 45.0, 47.5, and 55.0 as representative of potentials near the posterior ventricle is justified by the type and form of the P-wave resembling P_2 and on the similarity of QRS-T to that of the extreme left precordium. The intrinsic interval is approximately 0.04 seconds here, which is consistent with that found at points 5 and 6. Presumably some changes are also present in the left ventricle.

In summary, the major change of right ventricular hypertrophy is reflected into the exploratory leads of the right precordium, whereas the potentials from the anterior and posterior left ventricular regions resemble each other. This type of record should easily be differentiated from the electrocardiogram of anterior myocardial infarction.

Left Ventricular Hypertrophy

C. R., a colored man aged 79, had dyspnea on exertion, ease of fatigue, and general weakness for an indeterminate time before admission on October 12, 1941. No orthopnea or cyanosis; however, occasional edema of the right leg and a questionable anginal syndrome were present. The history was unreliable, as his memory was poor. Physical examination revealed moderate emphysema, fine moist rales over both bases, an apical systolic murmur without a thrill, occasional premature systoles, a bradycardia, variations in blood pressure between 170/100 and 196/90, and no evidence of ascites or enlarged liver. A two-metre X-ray film of the chest revealed marked enlargement of the heart, particularly of the left ventricle, as it appears to approach the lateral margins of the ribs. The costophrenic sinuses were hazy and were suggestive of congestion. No digitalis was given prior to the special study, and no rapidly progressive changes were noted in the electrocardiogram.

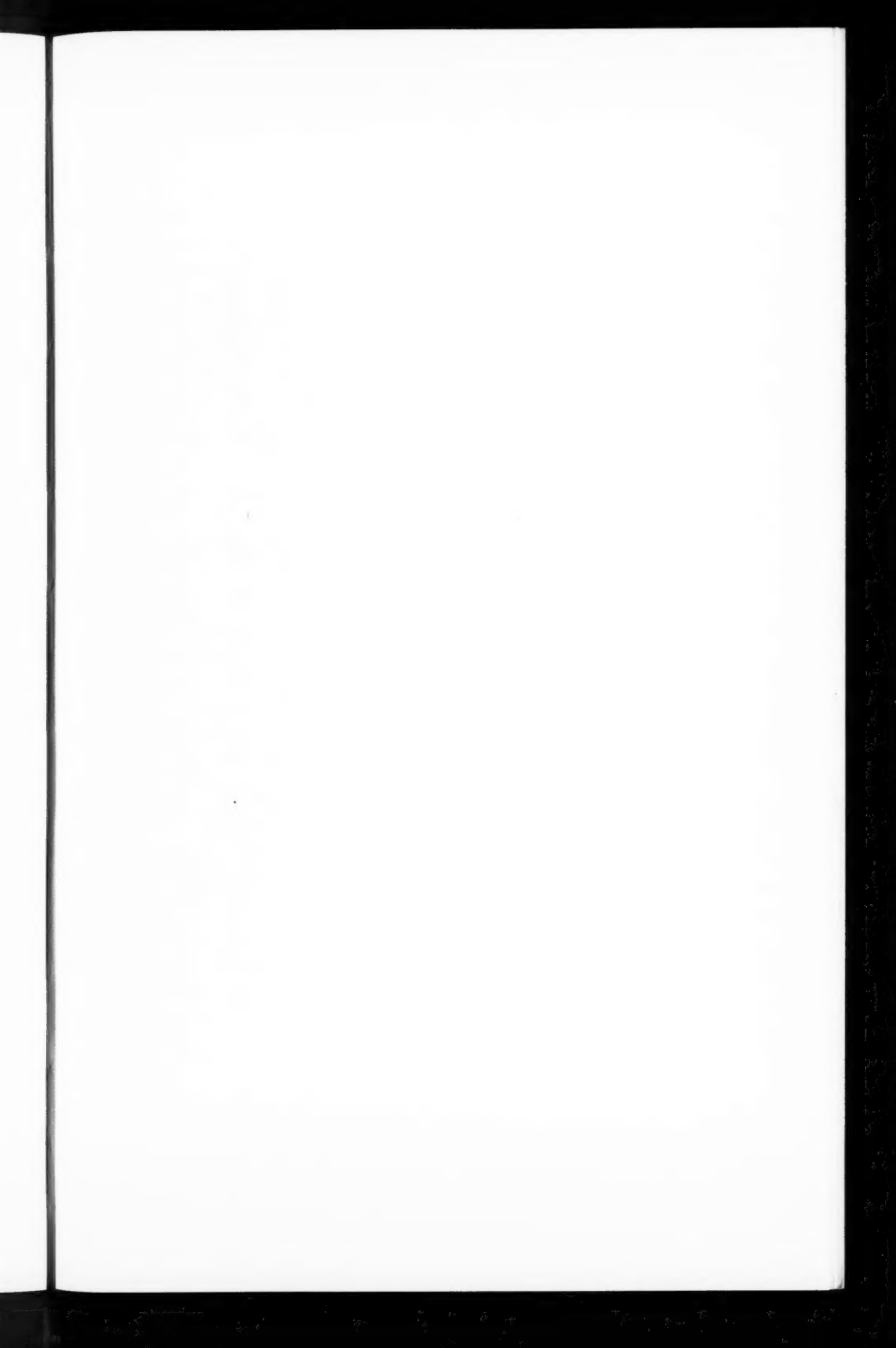
The standard electrocardiograms ($\frac{N}{T}$ sensitivity) on October 18, 1941 (Fig. 15), show a PR interval of 0.22 seconds and a QRS of 0.10 seconds as measured in Lead II. A tendency

toward left electrical axis deviation is present, as it measures $+5^\circ$ with the horizontal. High voltage is not definitely present. The inverted T-waves in Leads I and II are abnormal. These findings are not diagnostic of, but are found frequently in left ventricular hypertrophy of any etiology and sometimes in myocardial infarction of the anterior wall, and, therefore, require differentiation by exploratory leads (compare Fig. 3 and 4).

The single extremity potentials ($\frac{2N}{1}$ sensitivity) are of interest since the T-wave in lead RA is upright and abnormal, whereas the T-wave in lead LA is inverted (0.25 millivolts) more than normal and the T-wave in lead LL is nearly iso-electric. The QRS findings are within normal limits of Table I for these potentials.

The precordial potentials (0.6N sensitivity) show normal forms of QRS complexes from points 1 to 6. No delay in the intrinsic deflection is evident, as a fraction of the QRS complex is apparently iso-electric. The T-wave at point 3 is diphasic and abnormal. The T-wave at points 4, 5, and 6 is abnormally inverted up to 0.7 millivolts. These T-waves are located over the region of the anterior portion of the left ventricle. If similar changes are found posteriorly or in the stomach proximal to the ventricle, it would appear that a general change in the left ventricular muscle had occurred.

The esophageal potentials (0.6N sensitivity) show only abnormal forms with respect to T-waves at all levels, whereas QRS is essentially normal. Above the auricle at point 30.0 the T-wave is directed upward and the potential changes resemble those of lead RA. Proximal to the auricle at points 35.0, 40.0, and 42.5 the T-waves are abnormally electropositive. Points 45.0 and 50.0 are considered border-line in type between the auricle and the ventricle. Points 52.5, 55.0, 57.5, and 60.0, however, are considered proximal to the ventricle in the esophagus and stomach region. In these the QRS is of the normal type, but the T-waves are abnormally inverted to about 0.6 millivolts.



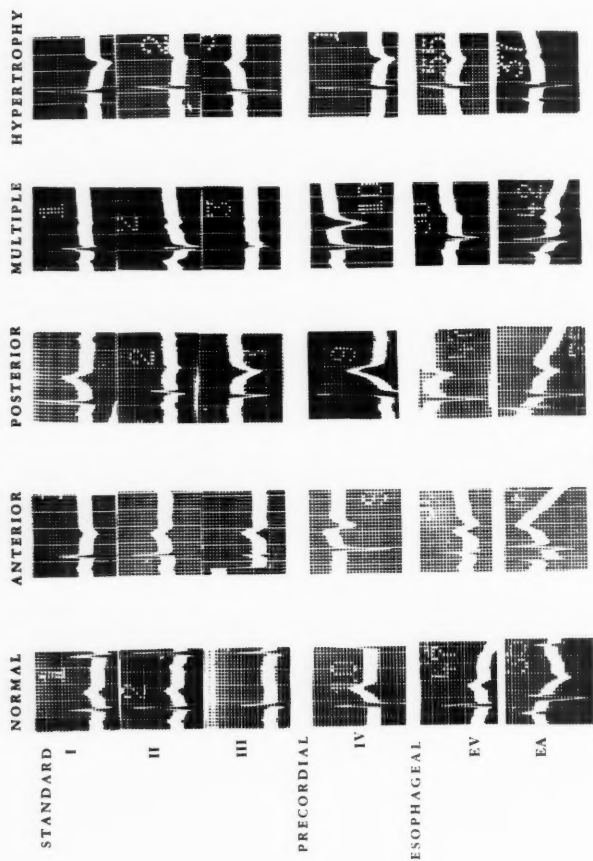


Fig. 16

This correlation of electrocardiograms demonstrates peculiarities of lead IV in anterior infarction, of lead EV in posterior infarction, of leads IV and EV in multiple infarction, and of leads IV and EV in left ventricular hypertrophy.

In summary, the potentials in the region of the left ventricle anteriorly and posteriorly reflect common changes with respect to T-waves, whereas QRS is of the normal type in each. This is interpreted as a result of left ventricular hypertrophy judging by the cardiac silhouette in the X-ray film. These changes are not a result of myocardial infarction in this case. Similar findings were described recently in the literature (8).

Comparative Study Using Selected Exploratory Leads
(Fig. 16)

A. *A normal man*, aged 26, revealed normal electrical axis, PR and QRS intervals, and normal form of QRS and T. Lead IV is normal with respect to P, QRS, and T at point 4 of the precordium. Lead E. V. shows a small Q in proportion to a normal R and S, whereas the T-wave is normally upright. The P-wave in IV is iso-electric, in E. V. it is smooth and upright, resembling P_1 and P_2 , and in E. A. it is sharply peaked and has a sharp (plus-minus) intrinsic deflection. The E. A. lead shows a normal deep Q-wave followed by a small R and an inverted T-wave.

B. *A case of anterior myocardial infarction* which was studied in detail in Fig. 3. The standard leads are of the atypical Q_1T_1 pattern. Lead IV reveals a deep Q-wave of 1.3 millivolts, no R deflections, and a diphasic (plus-minus) T-wave. This is diagnostic over the region of the infarction. Lead E. V. is normal. Lead E. A. shows an abnormal, upright T-wave. The precordial lead was of specific value in the diagnosis of this case.

C. *A case of posterior myocardial infarction* which was studied in detail in Fig. 5. The standard potentials reveal the typical Q_3T_3 pattern and give the diagnosis. Lead IV is of the normal type. Lead E. V. shows an abnormal, deep Q-wave, no R-wave, and a sharply inverted T-wave. This confirms and localizes the infarction in the posterior ventricular region. Lead E. A. is of the normal type; therefore, the E. V. lead and the standard leads were of specific value in the diagnosis of this case.

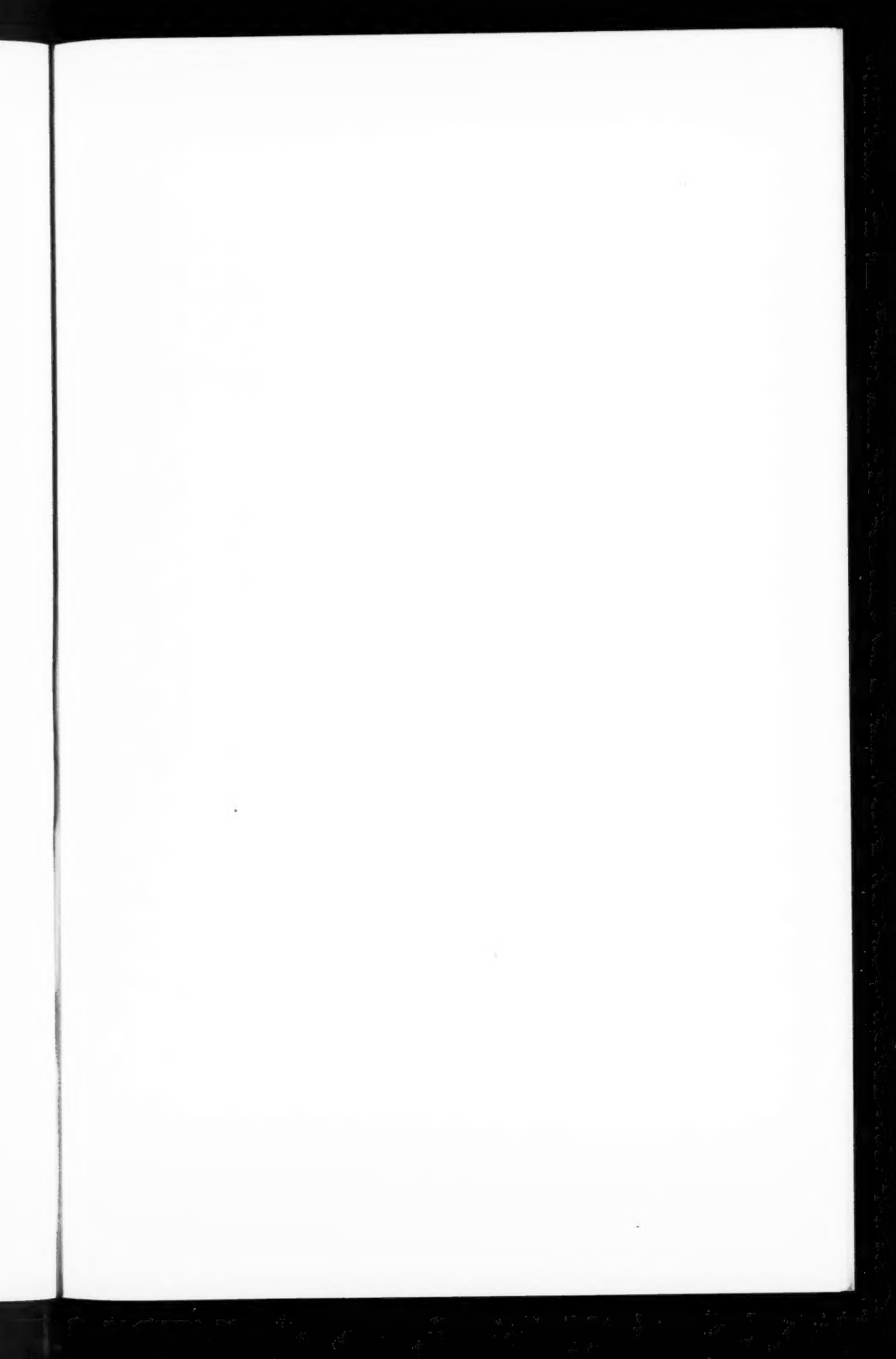
D. *A case of combined infarction of the anterior and posterior ventricular walls* which was studied in detail in Fig. 12b. The standard potentials are of the mixed QT pattern occasionally seen with myocardial infarction. Lead IV is abnormal, as a deep Q-wave of 1.5 millivolts, no R-wave, and a sharply inverted T-wave are present. This localizes a region of myocardial infarction over the ventricle anteriorly. Lead E. V. is also abnormal and composed of a deeply notched Q-wave and followed by an inverted T-wave. This localizes a region of infarction posteriorly. Lead E. A. shows an abnormally prominent R-wave and upright T-wave. In summary, Lead IV and lead E. V. were indicative of myocardial infarctions in the anterior and posterior walls and of great assistance in explaining the mixed QT pattern of the standard potentials.

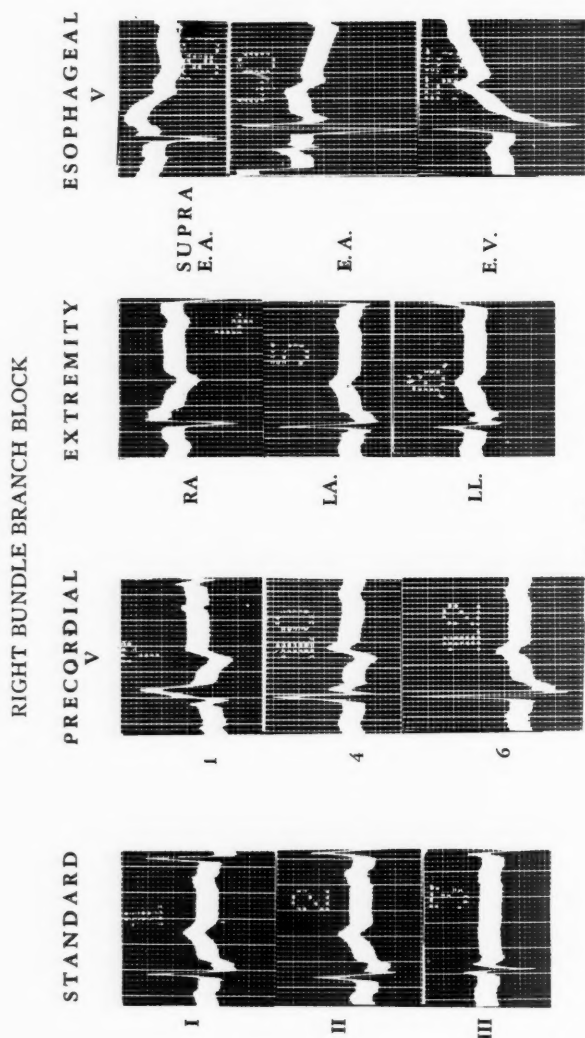
E. *A case of left ventricular hypertrophy* which was studied in more detail in Fig. 15 and reproduced here for comparison. The standard potentials show an electrical axis of $+5^\circ$, a PR interval of 0.22 seconds, and a QRS interval of 0.10 seconds. The T_1 and T_2 are abnormally inverted. Lead IV and lead E. V. reveal normal QRS complexes but abnormal inversion of T-waves. This bimural appearance of inverted T-waves is frequently found when a general change, such as hypertrophy, takes place in the left ventricle.

The distinctive differences under the above-named conditions appear evident from the studies of exploratory leads over the left ventricle using left precordial and esophageal ventricular leads. The electrocardiograms at the esophageal auricular levels are of no specific value in diagnosing ventricular myocardial infarction but reveal gross changes in right ventricular hypertrophy and bimural infarction.

Right Bundle Branch Block

M. H., a man aged 55, had a gradually progressive onset of symptoms of dyspnea and palpitation beginning about six months before entry to Bellevue Hospital on July 17, 1941. Ankle edema was present for three weeks. No knowledge of heart disease or





Right Bundle Branch Block is positively identified by leads from the right precordium showing delayed intrinsic deflection (RS).

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treatment of his condition existed. He was an unemployed chronic alcoholic with a history of an inadequate diet. On examination his blood pressure was 106/84 to 118/78; auricular fibrillation was confirmed by an electrocardiogram, at which time bundle branch block was also noted; the heart sounds were fair; and no murmurs were present. Evidence of decompensation disappeared on bed rest. No digitalis has been given since July 21. Persistent bundle branch block without significant change has existed for the past three months, although there was marked clinical improvement. Recently the heart has returned to regular sinus rhythm, seen in the special study. An X-ray film of the chest showed marked accentuation of the left ventricular curve, slight increase of the right ventricular shadow, and moderate increase in the transverse diameter of the aorta. The etiology of this disease was indeterminate; however, arteriosclerosis and malnutrition were considered most likely.

A standard electrocardiogram ($\frac{N}{I}$ sensitivity) on October 24, 1941 (Fig. 17), shows regular sinus rhythm, a PR interval of 0.20 seconds, a QRS interval of 0.16 seconds, and an electrical axis of 0° . A broad, slurred S_1 , S_2 , and M-shaped QRS_3 are present, while T_1 and T_2 are upright, and T_3 is iso-electric. These patterns do not strictly conform to any of the three types of right bundle branch block illustrated by the Criteria Committee of the New York Heart Association (15).

The single extremity potentials ($\frac{2N}{I}$ sensitivity) show a broad and slurred R-wave in RA, a broad and notched S-wave in LA, and LL, while the T-waves are normal in each lead. The direction and voltage of QRS in each lead are normal.

The precordial potentials ($\frac{N}{I}$ sensitivity) are typical of right bundle branch block as shown by Wilson and his collaborators (2,16). At points 1 and 2 the QRS is broad, notched, tall, and slurred. The downstroke of the major deflection (R) is referred to as the intrinsic deflection. It apparently occurs late in the cycle, which is about 0.12 seconds after the beginning of QRS in this lead. Normally the chief downstroke (RS) at these points

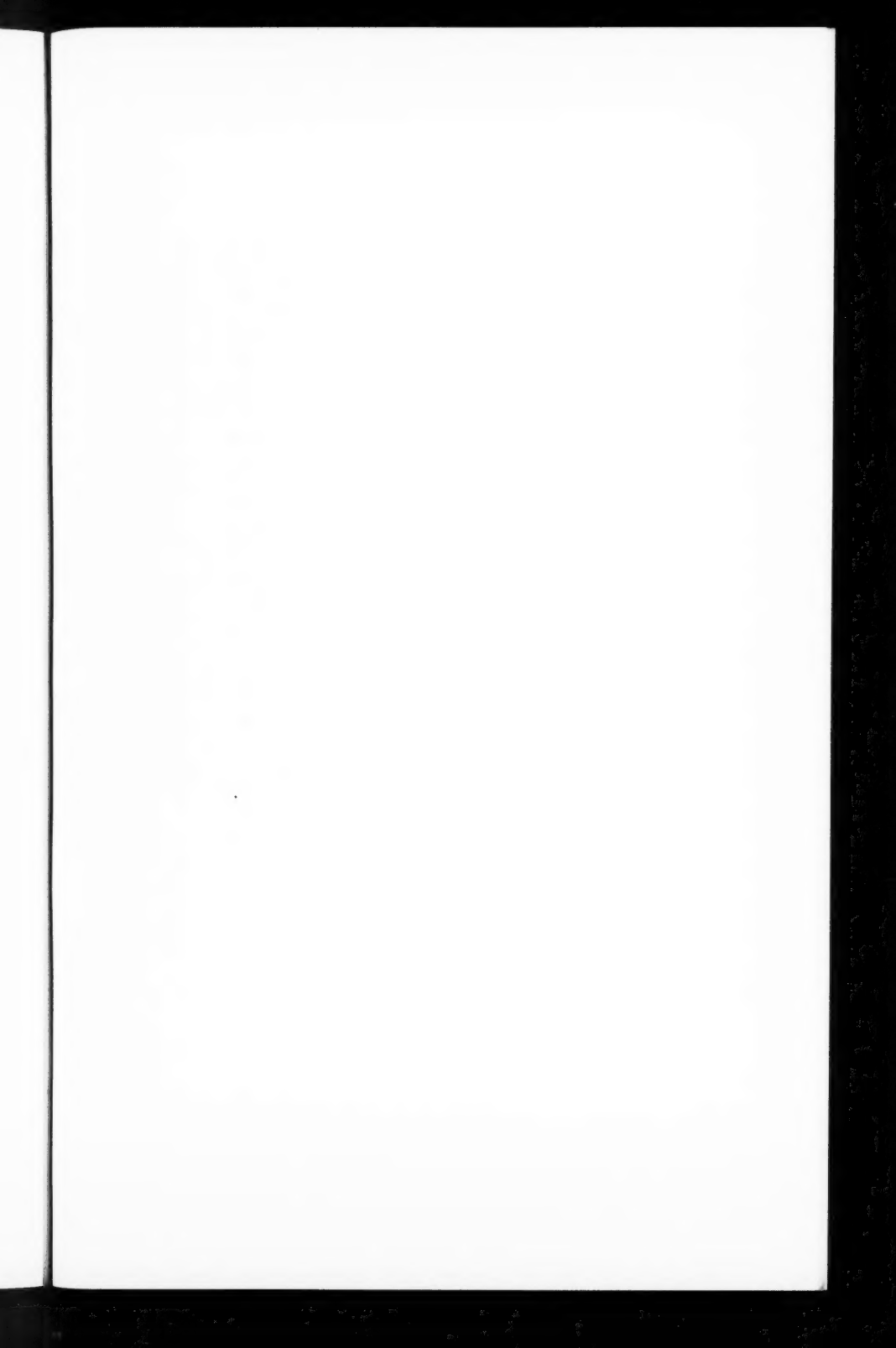
occurs in about 0.02 seconds after the earliest beginning of QRS in any lead (10,21). The T-waves are opposite in direction to the chief deflection over points 1, 2, 3, and 4. The form of the potential at point 4 is intermediate in type compared with those at points (1, 2, 3) and (5 and 6) in this case. At the latter point the intrinsic deflection (RS) begins in about 0.03 to 0.04 seconds after the beginning of QRS in this lead. This is normal for points over the left ventricle. The T-wave is directed upright in this region. Although the standard leads are atypical, the bundle branch block may be classified by the form of the precordial potentials at several points. In this case it is right bundle branch block.

The esophageal potentials ($\frac{N}{I}$ sensitivity) opposite the auricle (points 32.5, 37.5 and 42.5) show a slurring of the QRS complex late in the cycle, followed by an inverted or diphasic T-wave. The potentials at point 47.5 are intermediate in type. The potentials, however, proximal to the ventricle (points 52.5, 50.0, and 60.0) resemble those curves from the left precordium and left leg potential (LL) of this case. The intrinsic wave (RS) occurs normally in the QRS complex in about 0.03 to 0.04 seconds after the beginning of QRS in this lead, followed by a slurred S-wave and upright T-wave. It should be noted that opposite the auricle the intrinsic wave is present in the P-wave and not in the QRS complex.

In summary, it is apparent that the intrinsic deflection occurs normally over the left ventricle (anteriorly or posteriorly) and characteristically late over the right precordium in right bundle branch block.

Left Bundle Branch Block Concordant Pattern

J. M., a man aged 56, has never had any cardiac complaints; however, incidental to the study of an undiagnosed type of vascular disease of his lower extremities, an electrocardiogram revealed the presence of persistent left bundle branch block of the



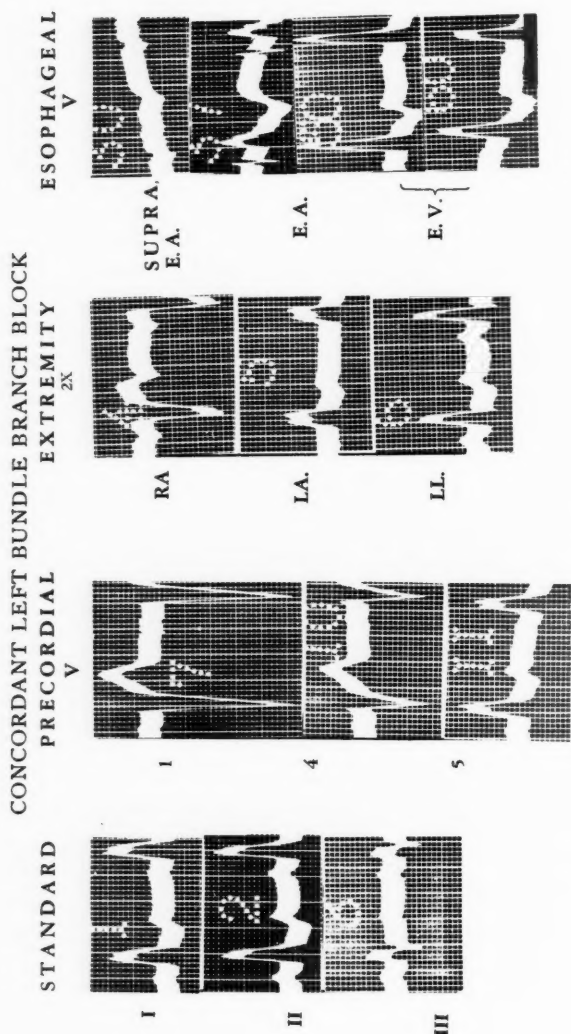


Fig. 18

Left Bundle Branch Block of the concordant type is identified by left precordial and esophageal ventricular leads, showing delayed excitation of these muscle areas.

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concordant type. A history of chancre was present in 1906, injection treatments for lues in 1911, and a recent Wassermann test was negative. The pulse rate was 76, the temperature 100° Fahrenheit, and the blood pressure 164/80 on admission to Bellevue Hospital December 16, 1941. The chest was of the rachitic type. The breath sounds were clear throughout, except for the accentuated aortic second sound. No other deviation from the normal heart was noted on auscultation and percussion.

The standard electrocardiogram ($\frac{N}{1}$ sensitivity) on December 20, 1941 (Fig. 18), shows a normal electrical axis of $+30^\circ$, a normal PR interval of 0.16 seconds, and an abnormal QRS interval of 0.16 seconds, which is consistent with a high grade of intraventricular block. Each QRS complex is broad and slurred, while in Lead III a small S-wave is present. The T-waves are mainly electronegative but diphasic in type. In general, the findings are also consistent with concordant left bundle branch block.

The single extremity potentials ($\frac{2N}{1}$ sensitivity) are of interest by comparison but contribute little to our knowledge at this time. Lead LL, however, resembles the form of precordial leads V_5 and V_6 , as well as that of the E. V. leads 50.0, 55.0, and 60.0 (Fig. 18).

The precordial potentials (0.65N sensitivity) show an early downstroke of the intrinsic deflection (RS) in potentials from V_1 to V_4 positions. The intrinsic interval measures about 0.02 seconds in this region, while the form of the complex is one comprised of small initial R-wave of less than 0.7 mv. and followed by a deep S-wave, reaching a maximum of 5.0 mv. in the V_2 region. These potentials are probably in the region of the myocardium supplied by normal impulses from the intact right Purkinje network.

On the other hand, in the region V_5 - V_6 the intrinsic deflection (RS) occurs late (0.08) in the QRS cycle and suggests delay of the arrival of the action current in the region of the left ventricle which is supplied by the left Purkinje fibers. If this is true generally in the left ventricle, the information obtained from esophageal leads should be of interest. The T-waves in V_5 - V_6 region are (minus-plus) diphasic in character and mainly inverted.

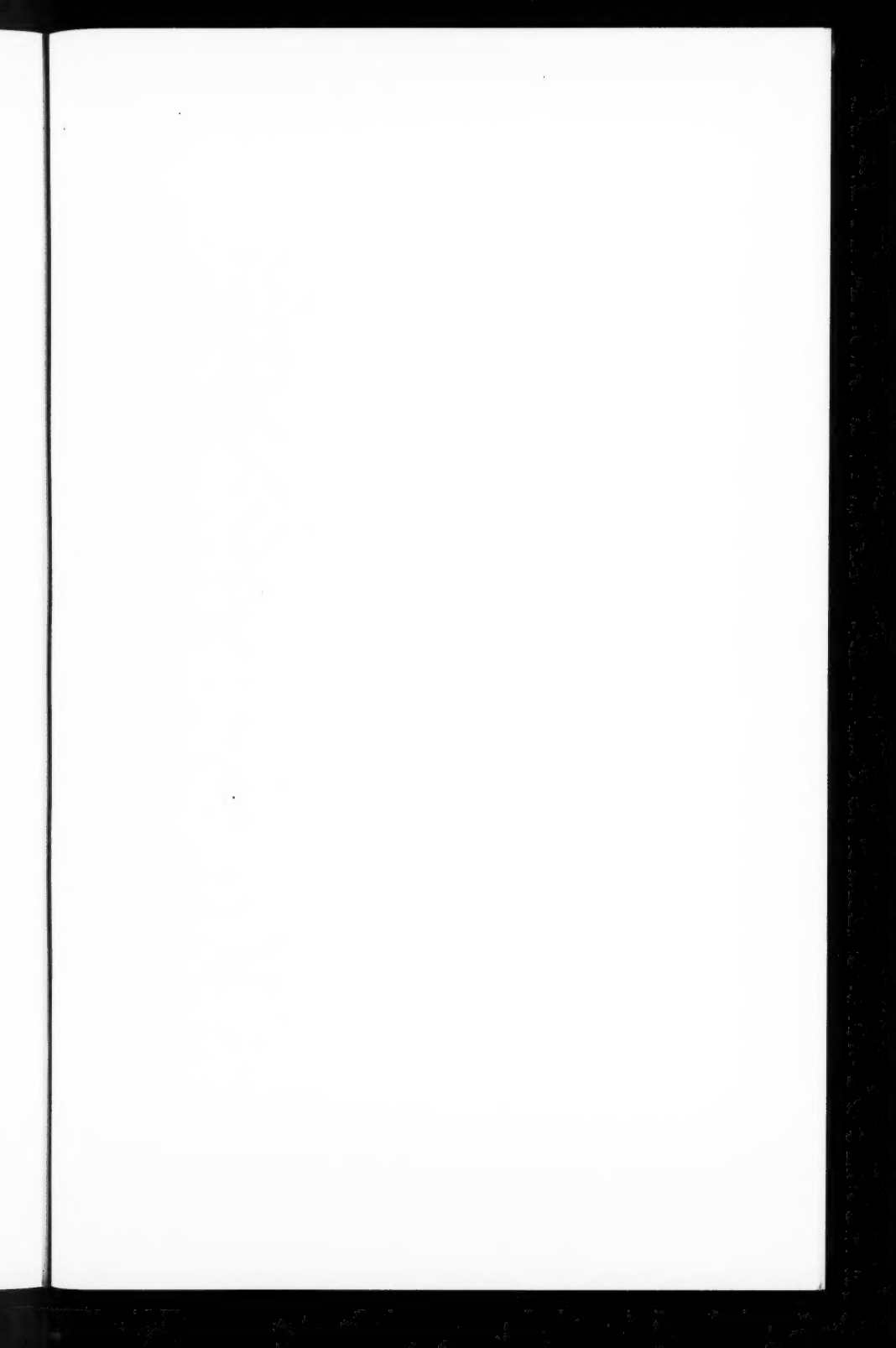
The esophageal potentials (0.65N sensitivity) at points 50.0, 55.0, and 60.0 are presumably in the region of the left ventricle posteriorly. In each the rapid downstroke of the intrinsic ventricular deflection (RS) occurs about 0.08 seconds after the beginning of QRS, and each is followed by an inverted T-wave. These QRS-T complexes in the E. V. leads closely resemble those from the left precordium (V_5 to V_6) and the foot potential (LL). These findings indicate that in some cases of left bundle branch block the action current is delayed in its arrival on the surface of the left ventricle but is normal in the region of the right ventricle. This supports the observations made previously on animals and man employing direct and indirect leads.

The potentials are abnormal in form and direction with respect to QRS and sometimes to T in the regions adjacent to the auricle at points 42.5, 40.0, 37.5, and 35.0. Above the auricle at point 30 QRS is small and bizarre, while the T-wave was normally inverted.

In general, we cannot assume that all cases of left bundle branch block have a similar form in anterior and posterior exploratory leads over the left ventricle in the absence of myocardial infarction. Recently (8) an exception to this was illustrated in a case of intraventricular block with a short PR interval. The following cases should be compared with the above (see Fig. 19a and 19b).

Left Bundle Branch Block Discordant Pattern

H. F., a male aged 70, was an out-patient in the cardiac clinic at Bellevue Hospital for a few years. His major complaint was gradual onset of dyspnea during the past six or seven years. Ten years ago, with his load as an iceman, he said he occasionally collapsed on climbing two flights of stairs. At present he suffers palpitation and vague precordial discomfort on climbing one flight. There was no history suggestive of coronary occlusion or frank decompensation. On physical examination an elevated blood pressure, general arteriosclerosis, and an enlarged heart



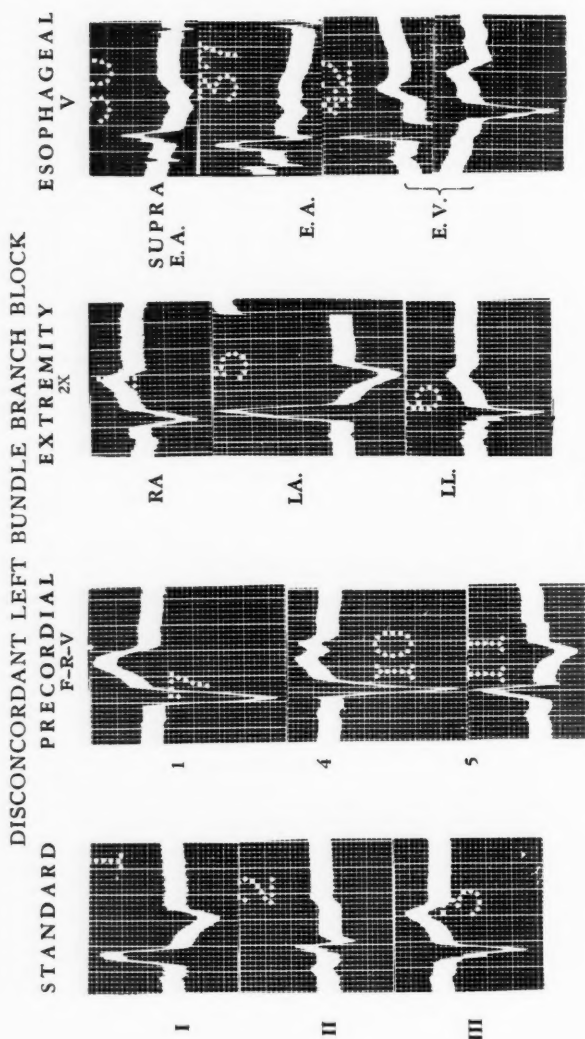


Fig. 19A

Left Bundle Branch Block of the discordant type is quite different from concordant. Although moderate left axis is present and leads III, LL, and EV are similar, it appears that the heart is rotated counterclockwise, as viewed from the apex. Thus EV and LL are dissimilar to precordial regions 5 and 6; however, they are similar to right precordial leads. They are not indicative of an infarct in these areas, but of early activation of muscle, probably the right ventricle.

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were found. No digitalis has been given. He was 5 feet, 6 inches in height and weighed 130 pounds. A left bundle branch block was demonstrated by an electrocardiogram. He was referred to the author for special studies.

The standard potentials (1.1N sensitivity) on December 13, 1941 (Fig. 19a), are consistent with left bundle branch block of the discordant type. The PR interval is normal and measures 0.16 seconds, while the QRS is prolonged and also measures 0.16 seconds. The Q-T interval measures 0.44 seconds, which is normal for a rate of 58 per minute. The shift in electrical axis to the left is equal to -38° . This may have effected an apparently deep Q_3 -wave of 1.8 mv., which is the only QRS_3 complex. There is no Q_2 deflection. T_1 is deeply inverted and opposite in direction to the chief QRS group. T_2 is low and diphasic; T_3 is tall and upright.

The unipolar extremity potentials (2.2N sensitivity), in general, are consistent with severe left electrical axis deviation in intraventricular block. The abnormal features are an upright T in lead RA, a high voltage R and deeply inverted T in lead LA, and a notched, deep Q-wave of 1.3 mv., which is the only QRS complex in lead LL. The T-wave in LL is normal and upright.

The precordial potentials (0.4N sensitivity) are similar to other published records (9,10,16) found in left bundle branch block. The QRS interval only measures 0.14 seconds at point 1, which suggests that part of the QRS is iso-electric in this complex, in which Q is the only QRS followed by a broad T-wave. At points 2, 3, and 4 the QRS is 0.16 seconds and the complexes begin with a small R of 0.1 mv. The intrinsic wave (RS) begins in about 0.02 seconds after the beginning of QRS at these same points. This value is normal for the arrival of the excitation wave over the right ventricle; however, it is abnormal for point 4 unless the apex is shifted to the left. Moderately notched T-waves at points 2, 3, and 4 may be related to the dissociation between right and left ventricles.

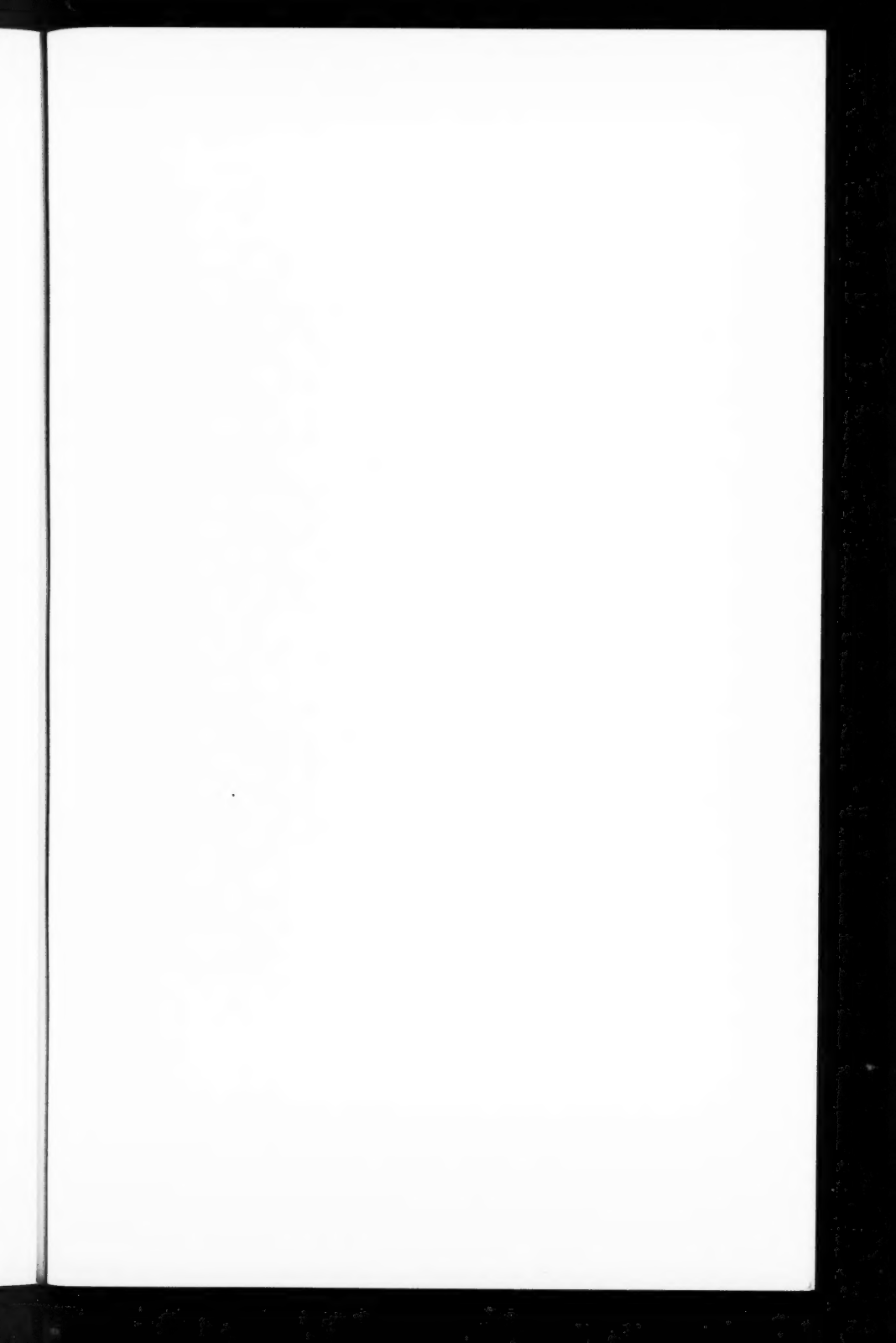
At points 5 and 6 the chief deflection is directed upward, as compared with points 1 to 4, where it is downward. The ventricular intrinsic wave (RS) occurs relatively late in the cycle and measures 0.08 seconds or greater, since part of QRS is iso-electric in each. By comparison with the above, the T-waves are abnormally inverted to 0.7 mv. These are localized to this region, since they are upright in the ventricular region posteriorly.

The esophageal potentials (1.1N sensitivity) are totally abnormal. Opposite the auricle, if we may judge by the diphasic P-waves, at points 30.0, 35.0 and 40.0 an initial R-wave appears to be the chief deflection of QRS followed by polyphasic T-waves at some positions. At points 45.0 and 47.5 (probably opposite the ventricle) an initial low voltage and slurred R-wave is present. The intrinsic deflection (RS) is about 0.06 seconds in a QRS complex measuring 0.14 seconds instead of 0.16 seconds. At these points the RS-T segments are slightly depressed and the T-waves (minus-plus) diphasic. In general, these potentials are neither like those of the right nor left precordium. At greater depths (points 55.0 and 60.0 in the stomach) apparently deep Q-waves of 1.5 mv. are present, which are slurred on the descending limb. These are followed by sharply upright T-waves. These findings are quite at variance with those of left bundle branch block with the concordant pattern (Fig. 18).

In summary, marked differences are apparently present in leads III, LL, and E. V. in the two typical forms of left bundle branch block; however, Wilson (47) suggests that a counter-clockwise rotation of the heart, viewed from the apex, may have brought a slip or area of the right ventricle in close proximity to the electrode, since the lower E. V. curves resemble the leads in the right precordium. Selective block of only a portion of the left bundle is not ruled out. This is highly improbable. Tall R in LA lead suggests a severe rotation to the left.

Left Bundle Branch Block Discordant Pattern

D. H., a man aged 64, on October 13, 1941, gave an unreliable history of paroxysmal dyspnea he called "asthma" for the



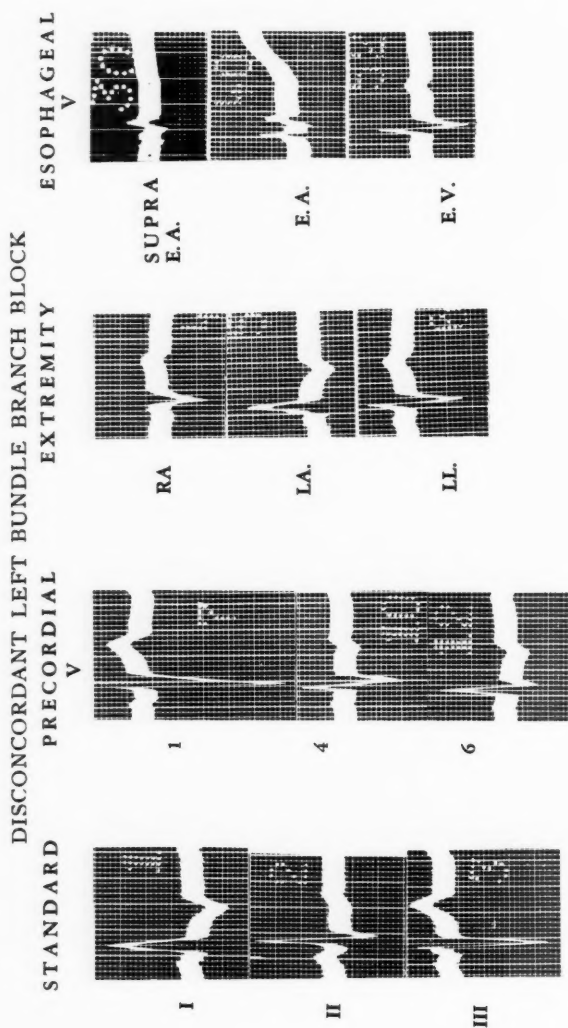


Fig. 19B

The patterns of discordant left Bundle Branch Block in this case are slightly different than observed in Fig. 19A.

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past two years. One day before entry he had a sudden attack of dyspnea not accompanied by precordial pain. On examination dyspnea at rest, enlarged heart with PMI in the anterior axillary line, moist rales at both lung bases, diaphragmatic breathing, traces of ankle edema, variable blood pressures from 138/72 to 168/72, a leukocytosis of 16,500, pulmonary congestion, and marked enlargement of the left ventricle by X-ray film were the positive findings. Electrocardiograms on October 13, 16, and 20 showed persistent left bundle branch block, discordant type, with inverted T_1 and T_4 , no evidence of progressive change, and no specific findings of myocardial infarction. On October 22 Fig. 19b shows:

The standard potentials ($\frac{N}{I}$ sensitivity) display the pattern of left bundle branch block of the discordant type described elsewhere (15). The PR interval is 0.14 seconds, the QRS interval is 0.12 seconds, the rate is about 68 per minute, and the electrical axis is -30° .

The single extremity potentials ($\frac{N}{I}$ sensitivity) show an abnormal, upright T-wave in RA, a prominent R in LA, and a deep S in LL. These findings are common in moderate to severe left axis deviation.

The precordial potentials (0.8N sensitivity) reveal an early intrinsic interval (about 0.02 seconds) over points 1, 2, and 3. At these points the S-waves are the chief QRS deflections, and the T-waves are upright. Point 4 is of the intermediate type. Points 5 and 6 are most characteristic of the left precordium and similar to those described for left bundle branch block (2). The intrinsic interval, as judged approximately in these leads, is relatively long (0.06 seconds). The R-wave is the chief QRS deflection, and the T-wave is inverted at points 5 and 6.

If both the anterior and posterior divisions of the left bundle branch are involved or if there was no rotation, it is likely that similar types of QRS-T complexes would appear in the esophageal ventricular leads. This, however, is not the case.

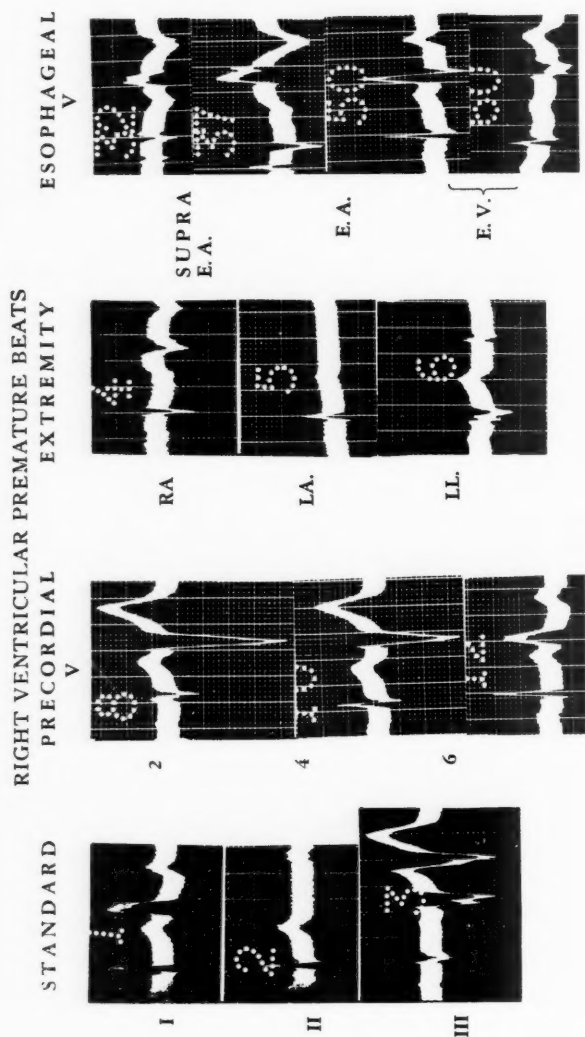


Fig. 20

Right ventricular premature beats coupled with a more normal beat in several of the leads simulate a pattern of Left Bundle Branch Block, discordant type.

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The esophageal potentials (0.35N sensitivity) show bizarre QRS complexes and nearly iso-electric T-waves proximal to the auricle at points 35.0, 37.5, and 40.0 and are difficult to evaluate. Proximal to the ventricle, however, at points 45.0, 50.0, 52.5, 55.0, and 57.5 the QRS is diphasic and the intrinsic deflection begins at 0.04 seconds, and the T-wave is upright. This interval is considered normal over the left ventricle; therefore, the posterior portion of the left bundle branch supplying this region may not be blocked. The etiology of this selective block may only be surmised at the present time, as in the previous cases (Fig. 18 and Fig. 19a). Wilson, however, suggests that the heart is rotated in a counter-clockwise direction (viewed from the apex). It is for this reason that esophageal leads from very low levels give semi-direct leads from the right ventricular surface and leads from higher levels, semi-direct from the left ventricular surface. When the standard leads are concordant, the heart is twisted the other way so that the left ventricle comes closer to the lower levels of the esophagus. This last explanation is probably correct.

Right Ventricular Premature Systole

M. A., a man aged 46, was in good health and an applicant for insurance on October 20, 1941. He has had frequent ventricular extrasystoles on examination for the past eight years. On physical examination, exercise, and fluoroscopy there was no evidence found of organic disease of the heart. As the extrasystoles appeared to be from a single focus, it was thought profitable to make a complete study of the form of the complexes in exploratory potentials obtained from the precordium and back of the heart.

In general, the initial normal complex resembles the normal form, in the standard, extremity, precordial, and esophageal electrocardiograms and need not be discussed further (Fig. 20).

The form of the premature systole in the standard leads ($\frac{N}{T}$ sensitivity) resembles the typical curve of left bundle branch block (discordant type, Fig. 19b). QRS_1 is a broad and slurred R-wave followed by an inverted T-wave. $QRS-T_2$ is intermediate

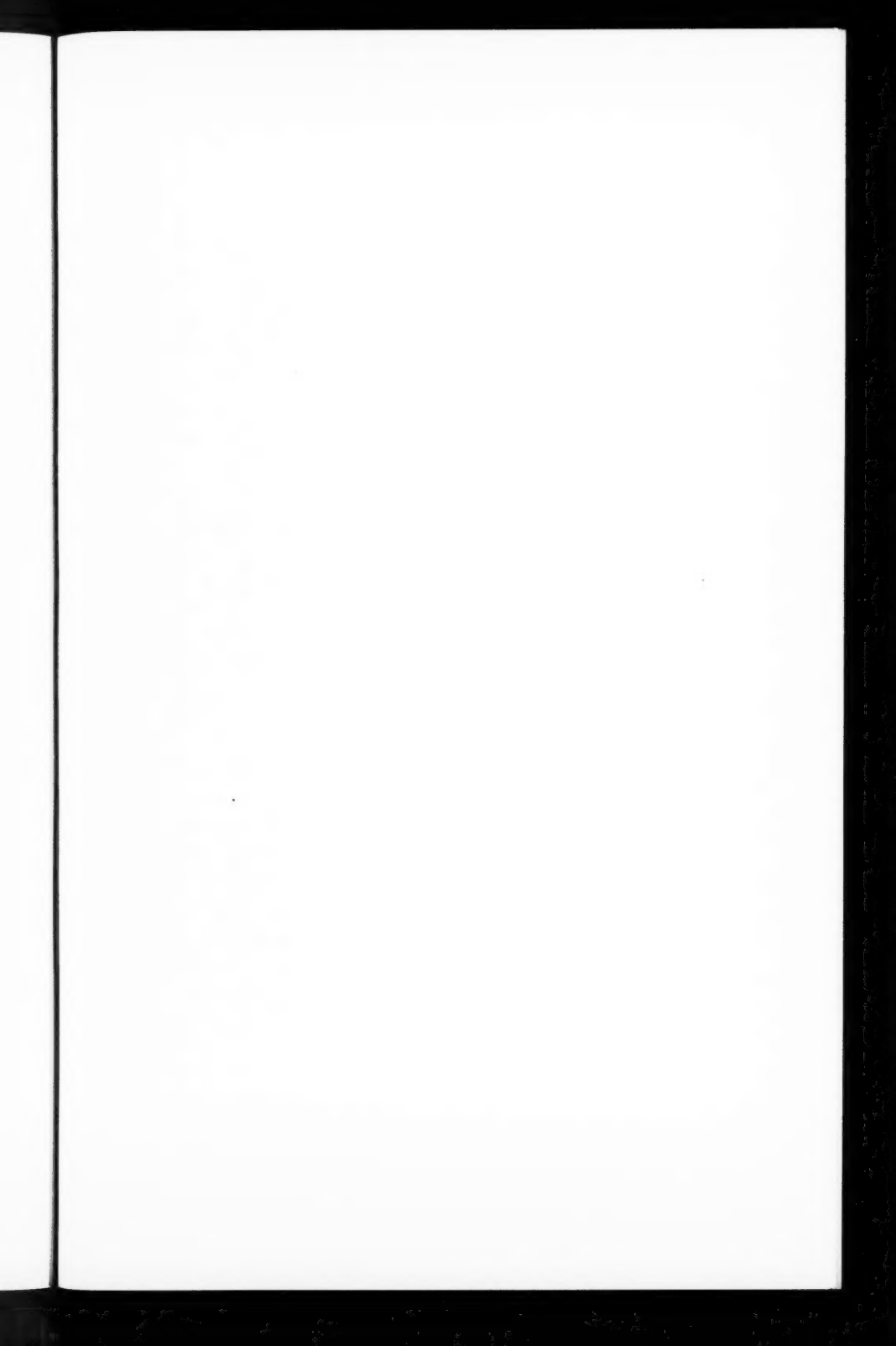
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in type. QRS_3 is largely composed of a broad and notched S-wave followed by an upright T-wave. The QRS complex has a 0.16-second interval and a left axis deviation of -13° . These complexes in the standard leads resemble those taken by Barker, et al. (23), following direct stimulation over the right ventricle in an exposed human heart.

The potentials as a result of premature ventricular systole in the single extremity leads occurred clearly only in lead LL and, therefore, will not be discussed.

The precordial potentials (0.7N sensitivity) of the premature ventricular beats show QRS complexes with an early intrinsic deflection (RS) and occur on the right precordium at points 1, 2, and 3. These are followed by tall, upright T-waves. Point 4 is intermediate in this case. Points 5 and 6 on the extreme left precordium show a major deflection (R) which is slurred and upright. The rapid downstroke of R occurs late (about 0.07 seconds) in the QRS cycle, which is followed by a lower upright T-wave than at points 1, 2, and 3. These findings are consistent with the hypothesis that this premature beat excites the right ventricle first. Then, the action current progresses through the muscle across the septum to the left myocardium, if we may judge by the delayed intrinsic deflection in this region.

The esophageal potentials (0.6N sensitivity) of the premature ventricular beats opposite the auricle at points 32.0, 37.5, and 40.0 show a major R-wave followed by an inverted T-wave and a superimposed polyphasic P-wave containing the intrinsic auricular deflection. Judging by the form of the P-wave at points 45.0, 47.5, 50.0, 52.5, 55.0, 57.5, and 60.0, the potentials are proximal to the ventricle in the esophagus or stomach. In each, except at point 60.0, the R-wave is the major deflection of QRS, and it is followed by an upright T-wave. Premature beat at point 60.0 resembles curves recorded from the right precordium and ventricle. The intrinsic deflection (RS) tends to occur late (about 0.06 seconds at points 52.5 and 55.0) in this region. Since the intrinsic interval is delayed over the left ventricular region



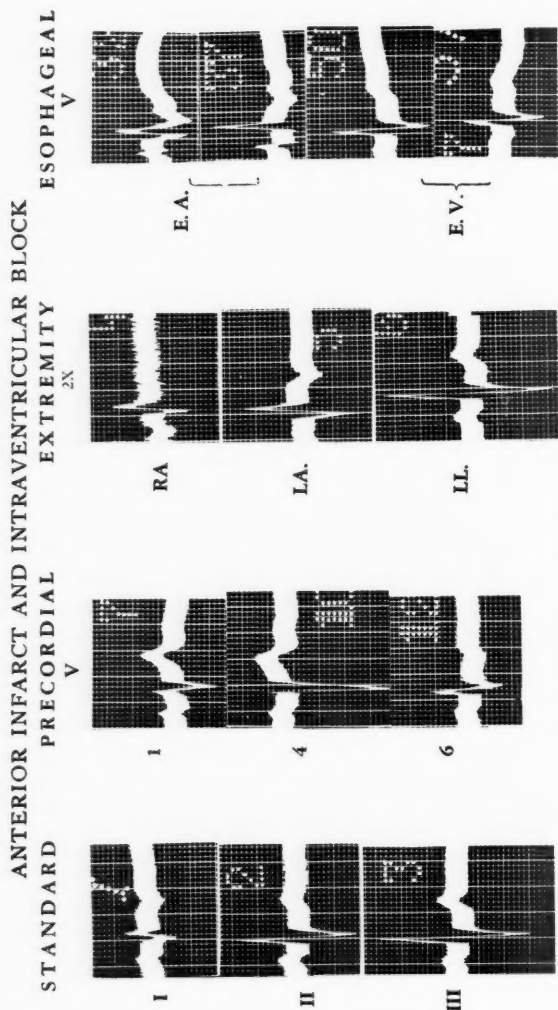


Fig. 21

Identification of infarcts in the presence of intraventricular block is possible on the anterior wall of this case because R is distinctly smaller in region 4 than in regions 1 and 6. A left or right preponderance is not shown here. The abnormal deep Q in LA lead is often seen in anterior myocardial infarction. This is not specific for this condition.

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(anteriorly and posteriorly) and is early over the right ventricle, it is easily deduced that the premature systolic impulse originated in the right ventricle of this case.

In general, the form of the ventricular complex in the exploratory leads of the precordium and esophagus is helpful in diagnosing the site of the new impulse formation. This is an instance of a single focus arising in the right ventricle. The resultant form of the complexes in the premature beat in the several leads is consistent with those predicted in typical and complete left bundle branch block of the discordant type.

Intraventricular Block and Probable Anterior Myocardial Infarction

B. G., a man aged 58, was first admitted to Bellevue Hospital because of severe precordial pain on November 15, 1940. He had no cardiac symptoms or knowledge of heart disease prior to this time. On examination he was acutely ill and in cold sweat. His pulse was regular, feeble, and 100 per minute. The heart sounds were faint. His blood pressure was 106/88 on admission and 90/62 one month later when he experienced further chest pain. A precordial friction rub was heard at this time. There were no signs of cardiac decompensation. An X-ray film on December 2, 1940, showed moderate accentuation of the left ventricular curve and widening of the supra-cardiac aorta. The electrocardiograms revealed progressive changes conclusive of infarction on the anterior myocardium. Later he had two admissions on June 27, 1941, and on October 15, 1941, because of a constant, severe anginal syndrome not relieved by nitroglycerine. A nerve block was ineffective in relief of this pain. In the out-patient clinic he was given digitalis on evidence of decompensation. A grain and one-half per day was taken routinely for several weeks until the present electrocardiographic study in Fig. 21.

The standard electrocardiograms ($\frac{N}{T}$ sensitivity) show low voltage of QRS_1 . Q_1 is 0.25 millivolts and equal to R_1 . T_1 is moderately inverted. Deep S_2 and S_3 and upright T_2 and T_3 are present. The minor deviations in RS-T segments are difficult

to evaluate in the presence of digitalis. The PR interval is normal. The QRS interval is prolonged to 0.12 seconds, which is indicative of intraventricular block. These findings are suggestive of the anterior type of myocardial infarction.

The single extremity potentials ($\frac{2N}{1}$ sensitivity) show an abnormal, iso-electric T in lead RA, and abnormal, deep Q-wave in LA, and elevated RS-T segments in RA and LA. These are not conclusive of infarction; however, these are indicative of myocardial deviations from the normal.

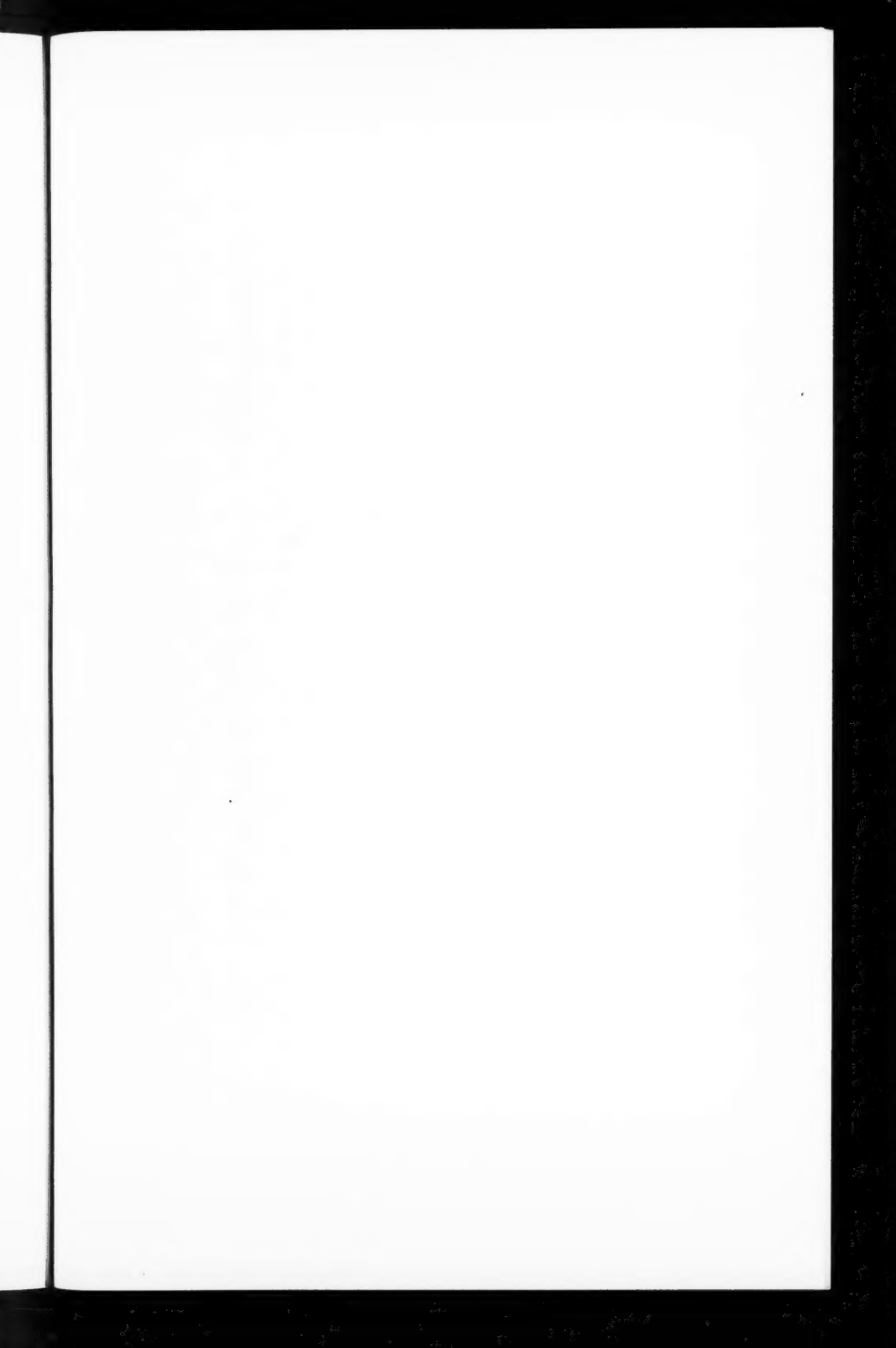
The precordial potentials ($\frac{N}{1}$ sensitivity) are essentially normal at points 1 and 2. The initial R-wave becomes smaller instead of taller at points 3, 4, and 5 and is less than 0.2 millivolts in each record. The T-waves are abnormally inverted at points 5 and 6. These findings, with the history, are adequate to establish a diagnosis of anterior myocardial pathology in spite of an associated intraventricular block.

The esophageal potentials ($\frac{N}{2}$ sensitivity) are abnormal proximal to the auricle at points 35.0, 37.5, and 40.0, since the R-wave is the major QRS deflection and the T-waves are upright or diphasic. Superimposed mechanical artefacts obscure the true character of the T-waves. Point 42.5 is considered at the margin of the auricle and ventricle. Potentials at points 45.0, 50.0, 55.0, and 57.5 are considered proximal to the ventricle and are of the normal type. This was expected in the chronic stage of anterior myocardial infarction.

Intraventricular Block

Probable Bimural Myocardial Infarction

K. O., a Japanese male aged 45, had heart burn, and epigastric and lower abdominal distress as a result of gas for the past five years. For the past three months he complained of shortness of breath, orthopnea, moderate swelling of the legs, and for two months a slightly productive cough. He worked as a cook until three weeks before entry. Since digitalis aggravated his symptoms he discontinued it (one tablet, O. D.) a few days before entry



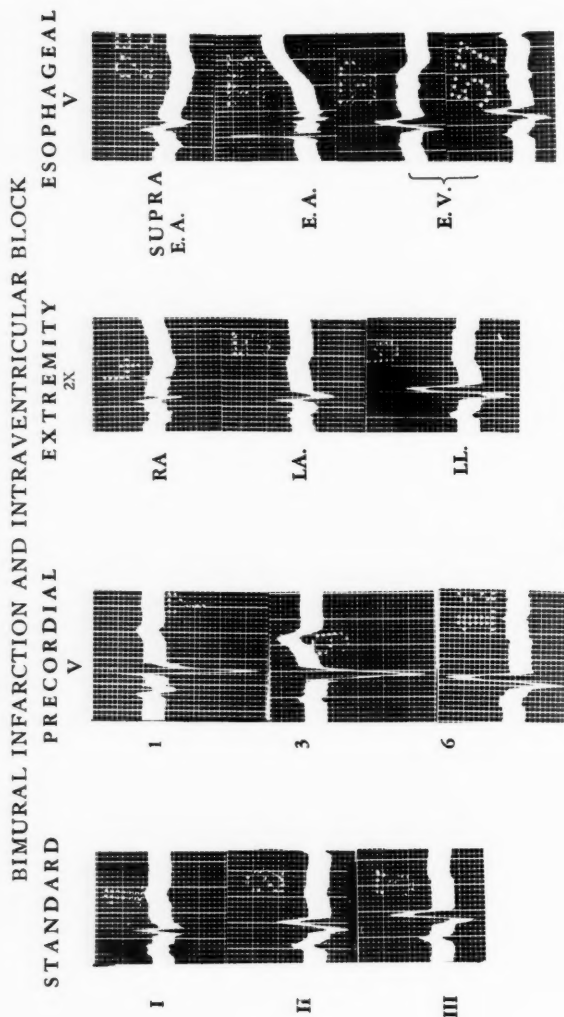


Fig. 22

It is usually stated that infarcts are easily identified when right Bundle Branch Block is present, but extremely difficult in left Bundle Branch Block. In this case, judging by the late downstroke of RS in region 6 and EV, we may be dealing with left Bundle Branch Block of the concordant type. Judging by deep Q in regions 3, 6, and EV, we are dealing with bimural infarction. These multiple lesions are the basis for the mixed Q-T patterns of the standard leads. The initial R is prominent in RA, and Q is prominent in LL.

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to the hospital on October 15, 1941. His cardiac status summarized on December 11 on transfer to a chronic hospital was that of arteriosclerotic heart disease with an enlarged heart, probable myocardial infarction, myocardial failure, enlarged liver, pulmonary emphysema, regular sinus rhythm, and Class IV E. His blood pressure was 128/90, pulse rate 84 per minute, respiration rate 20 per minute, and erythrocyte sedimentation rate elevated (24-36 mm. per hour). The following study was made when the subject was essentially free of digitalis.

The standard electrocardiogram ($\frac{N}{I}$ sensitivity) of October 25, 1941 (Fig. 22) shows regular sinus rhythm, a normal PR interval of 0.16 seconds, a prolonged QRS interval of 0.12 seconds, and a normal electrical axis. The form of QRS is abnormal in each standard lead. QRS_1 is of low voltage and W-shaped. It is followed by an upright T-wave. QRS_2 is initiated by an abnormal, deep Q-wave of 0.3 mv., which is one-half of R_2 voltage. QRS_3 is also initiated by a deep Q-wave of 0.35 mv., which is 50% of R_3 voltage in this case. T_3 is inverted but of low voltage and followed by a prominent, upright U-wave. In general, the data are non-specific for bundle branch block of a given type and for myocardial infarction, if we may judge by the form of QRS-T in each lead.

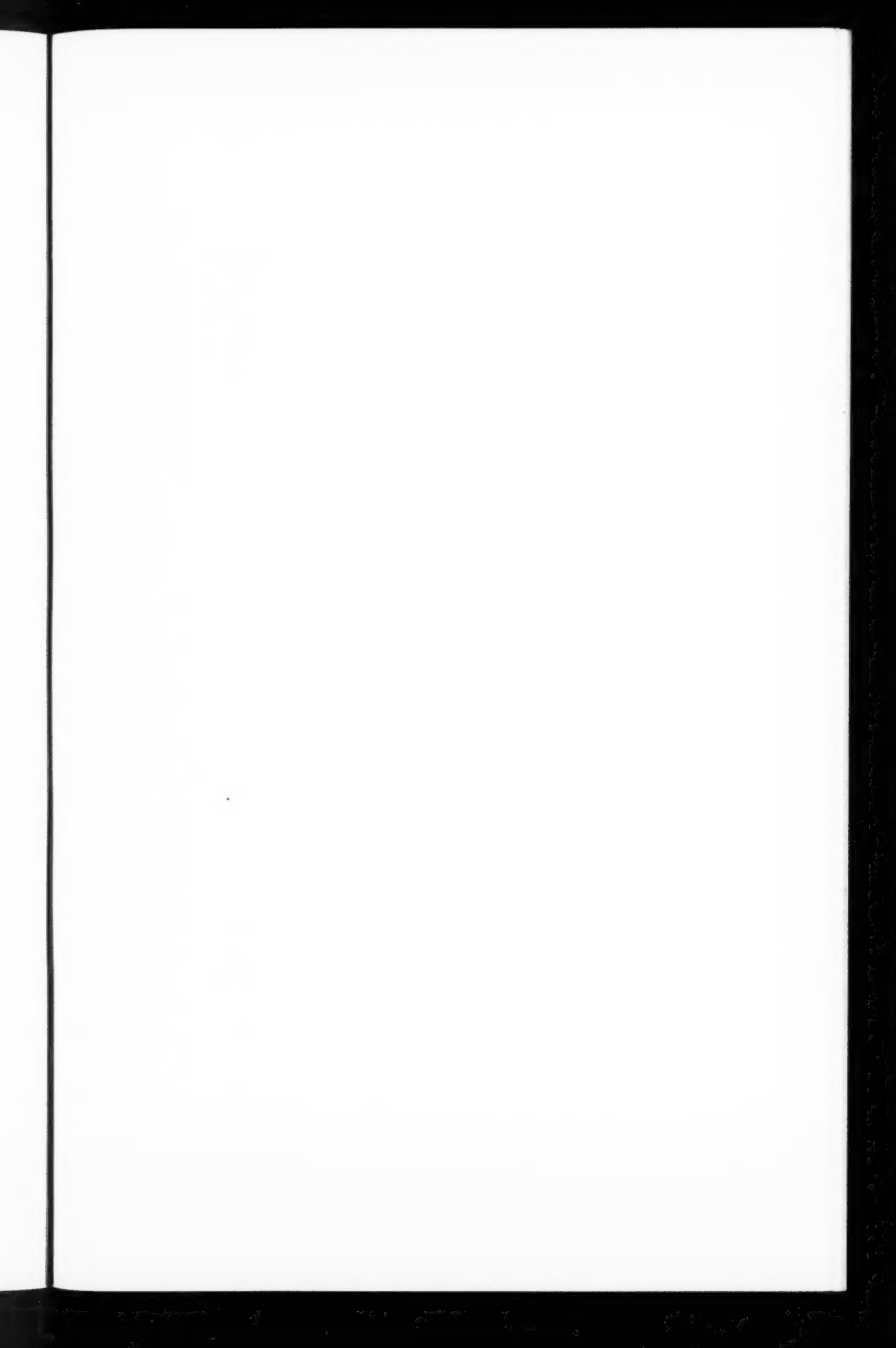
The unipolar extremity potentials ($\frac{2.1N}{I}$ sensitivity) reveal a low voltage, M-shaped QRS complex in lead RA; however, a normally inverted T-wave. The form of QRS-T in lead LA is within normal limits; however, an abnormal Q-wave of 0.25 mv. is present in lead LL. This finding has often been observed concomitant with evidence of posterior myocardial infarction in other leads.

The precordial potentials ($\frac{1.2N}{I}$ sensitivity) appear normal except for a wide QRS at point 1. At points 2 to 6 the QRS deviates from normal. At point 2 there is a suggestion of an initial Q-wave and a very prominent U-wave. At point 3 a deep Q-wave of 2.0 mv. is present; however, it is notched on the early descending limb. At point 4 the Q-wave is less marked but in-

creases in magnitude to 0.5 mv. at point 6, which is probably significant. These deviations in QRS over the mid- and left precordium are suggestive of the presence of an anterior or lateral myocardial infarct. Judging by the late (0.08 seconds, maximum) rapid downstroke of the intrinsic wave (RS) at points 5 and 6, we are probably dealing with a left bundle branch block defect. The T-waves are normal at points 1 to 5, but at point 6 the T-wave is slightly inverted. Unfortunately, in the presence of a prolonged QRS interval the direction and character of T is difficult to evaluate in terms of myocardial change.

The esophageal potentials ($\frac{N}{T}$ sensitivity) were difficult to record without artefacts occurring in the T-waves but were of some assistance in this case. Point 25.0 is above the region of the auricle and resembles potential RA. Points 32.5, 37.5, and 40.0 are proximal to the auricle and reveal a broad, notched, and bizarre QRS complex, which is mainly electropositive and followed by a slightly negative T-wave. Point 42.5 is border-line in position. Points 45.0, 47.5, 52.5, 57.5, and 60.0 are recorded from the ventricular region. The presence of Q-waves measuring 0.4 mv. (maximum) which are almost equivalent to the succeeding R-waves is probably significant and confirms our suspicion of posterior wall pathology suggested in the standard leads. The T-waves in each case are of low voltage and polyphasic. In general, the E. V. potentials resemble the potentials at point 6 on the precordium and those of the left leg. The apparent intrinsic wave (RS) in the E. V. potentials occurs about 0.08 seconds after the beginning of QRS. This is further support that a bundle branch conduction defect is present in the left ventricle, whereas the intrinsic interval over the right ventricle measures less than 0.02 seconds at points 1 to 3, which is normal for these positions.

In summary, judging by the complex electrocardiogram in the several leads, we are probably dealing with a concordant type of left bundle branch block in the presence of myocardial infarction involving the posterior and lateral or anterior myocardial walls of the ventricles. (Compare Fig. 18 and Fig. 11a, 11b.)



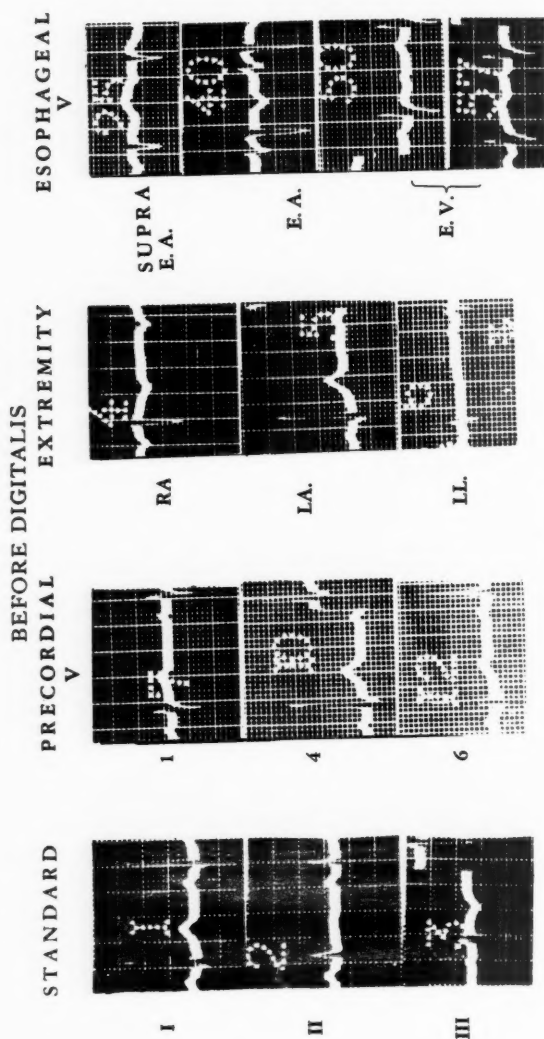


Fig. 23A

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The Effect of Digitalis

G. A., aged 54, an active gardener, has had a gradual onset of and progressive increase in dyspnea on slight exertion which he antedates about thirty years, when he had three attacks of pneumonia and one of pleurisy. His blood pressure was about 122/78, and his heart rate was about 75 per minute at rest but much faster during slight exertion and emotion. The rate was not appreciably decreased during digitalis therapy but adequate to produce electrocardiographic changes. On fluoroscopy there was slight prominence of the left ventricle, while the base of the left lung was very hazy, no fluid level being demonstrable. Basal pulmonary rales were usually present on auscultation, especially at the left base, where impaired resonance and moderately diminished breath sounds were also found.

Before Digitalization (Fig. 23a)

The standard leads ($\frac{N}{I}$ sensitivity) show (Fig. 23a) a rate of 78 per minute, a PR interval of 0.18 seconds, a QRS interval of 0.08 seconds, a Q-T interval of 0.4 seconds, and an electrical axis of $+5^\circ$. QRS₂ is low voltage, and T₂ is nearly at isopotential, which is abnormal.

The single extremity potentials (1.8N sensitivity) were consistent with left axis deviation as judged by QRS in lead LA. QRS is bizarre, and T may be abnormally inverted in LL.

The precordial potentials ($\frac{N}{I}$ sensitivity) are normal at points 1 to 6 with respect to QRS, and at points 1 to 4 with respect to T; however, the T-wave of 0.1 mv. at points 5 and 6 is considered abnormally low.

The esophageal potential ($\frac{N}{I}$ sensitivity) at point 25.0 is considered normal and above the auricle. Points 32.5, 37.5, 40.0, and 42.5 are probably adjacent to the auricle and normal. Point 47.5 is border-line in type, whereas points 50.0, 55.0, and 57.5 are in the region of the ventricle. The QRS is diphasic in type, resembling points 1 and 2 on the precordium, whereas the T-waves are low and upright, about 0.1 mv., resembling those from points 5 and 6.

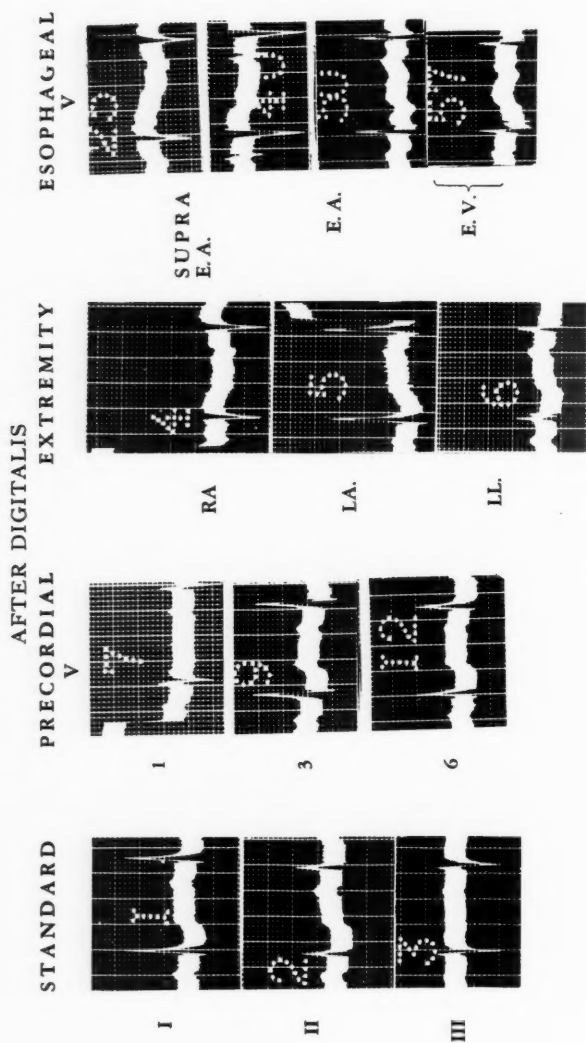


Fig. 23B

Special studies of a subject with left axis deviation before and after toxic doses of digitalis reveal interesting changes in the RS-T segment. The inverted T wave in lead LL may be abnormal prior to digitalis administration. Tall R in LA is characteristic of left electrical axis deviation.

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After Digitalization (Fig. 23b) (Sensitivity about the same as in previous study for a given lead.)

The PR interval and QRS are unchanged; however, the Q-T interval shortens to about 0.30 seconds as compared with 0.40 seconds before digitalization. The P-wave and QRS complex are unchanged by the drug in all the leads.

The RS-T segments become depressed in Leads I, II, LA, LL, V₂, V₃, V₄, V₅, V₆, and at esophageal points 47.5, 50.0, 52.5, 55.0, and 57.5 near the ventricle. Elevated RS-T segments are noted in lead RA, above the auricle (25.0), and opposite the auricle at points 32.5, 37.5, 40.0, and 42.5. Iso-electric RS-T segments are noted in Lead III and Lead V₁.

The T-waves are especially lowered in voltage in Lead I, II, LA, V₁ to V₆, and at points 50.0 and 52.5 as a result of the medication.

All these findings are consistent with the toxic effect of digitalis produced on the right and left ventricular myocardium described previously by standard and precordial leads. The case illustrates the completeness with which digitalis modifies the electrocardiograms in exploratory leads anteriorly and posteriorly.

Non-Specific Posterior Myocardial Changes and Intraventricular Extrasystoles

T. F. R., a male aged 34, physician, applied for \$3,600 Retirement Income at Age 55. His personal history reveals hemolytic streptococcal infection of the nasal sinuses three years previously. He suffered a fractured right arm in a train accident one year prior. This healed following operative intervention. Examination revealed a normal build, blood pressure, and pulse. A soft apical murmur was present with transmission to the axilla on exercise. There was no cardiac enlargement or rheumatic fever history.

On comparison of June 22, 1943, and July 8, 1943, electrocardiograms, we find abnormally progressive changes of T₂ and T₃ and occasional unclassified ventricular premature beats.

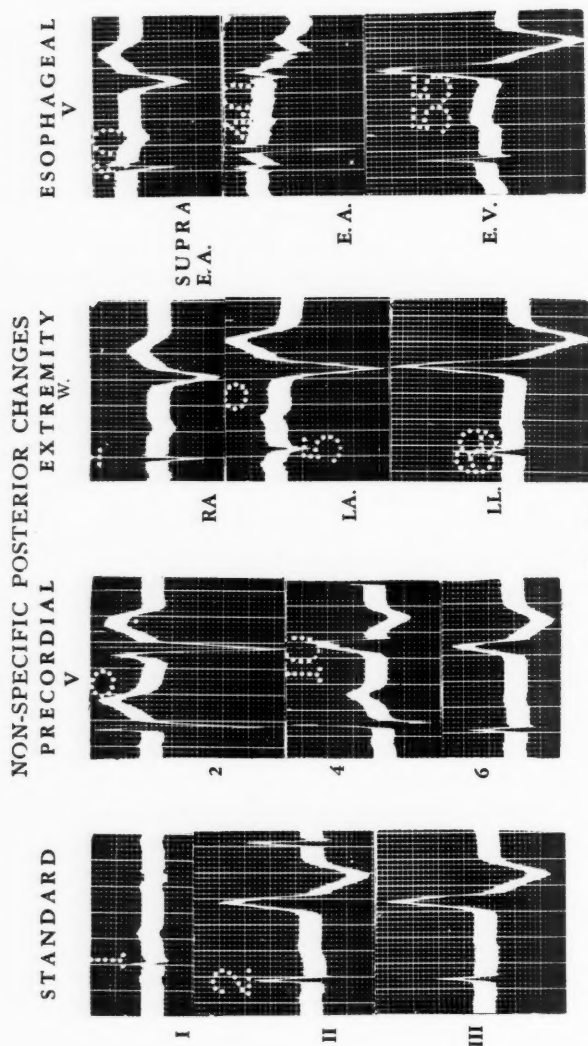


Fig. 24

Special study of ventricular extrasystoles probably arising high in the ventricular septal region. There are also localized non-specific changes involving the posterior wall, if we may judge from the peculiarities of the initial beat in the esophageal ventricular lead.

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On study at the Home Office, an inconstant, faint systolic murmur was noted at the apex; however, it increased and was constant after exertion. Exercise tolerance was normal. Fluoroscopy showed a normal vertical heart. The electrocardiogram (August 26, 1943) showed many interesting changes.

The standard leads show a low voltage QRS_1 and normal T_1 of 1 mm., a border-line diphasic T_2 , and an inverted T_3 of 1 mm. The ventricular extrasystoles tend to follow the general pattern of the initial normal beat except for the wide QRS interval. There were no T-wave changes in the following beats.

The unipolar extremity potentials favor a slight right axis shift. The T-wave in LL is slightly multiphasic and essentially iso-electric. The ventricular complexes of the premature beats are again similar in form to the preceding beat; however, T_{LA} is upright and T_{LL} is inverted and related to large QRS area in the opposite direction.

The precordial series are normal with respect to the initial beat in V_{1-6} region. The ventricular extrasystoles show a delayed intrinsic (RS) wave over the right and left portions, suggesting a high intraventricular or septal focus.

The esophageal series are locally abnormal with respect to T at all ventricular levels (50.0, 52.5, and 55.0) and normal above this. The premature beats also show delay in the intrinsic wave (RS), leading to the conclusion that the beat probably arises high in the septum and then distributes itself along the same electrical course as the Purkinje network. This is deduced from its similarity to the preceding beat.

In conclusion, it was shown that the changes in T_2 and T_3 are reflected into an abnormality only localized by the esophageal ventricular leads. The premature beat probably originates in the high septal region.

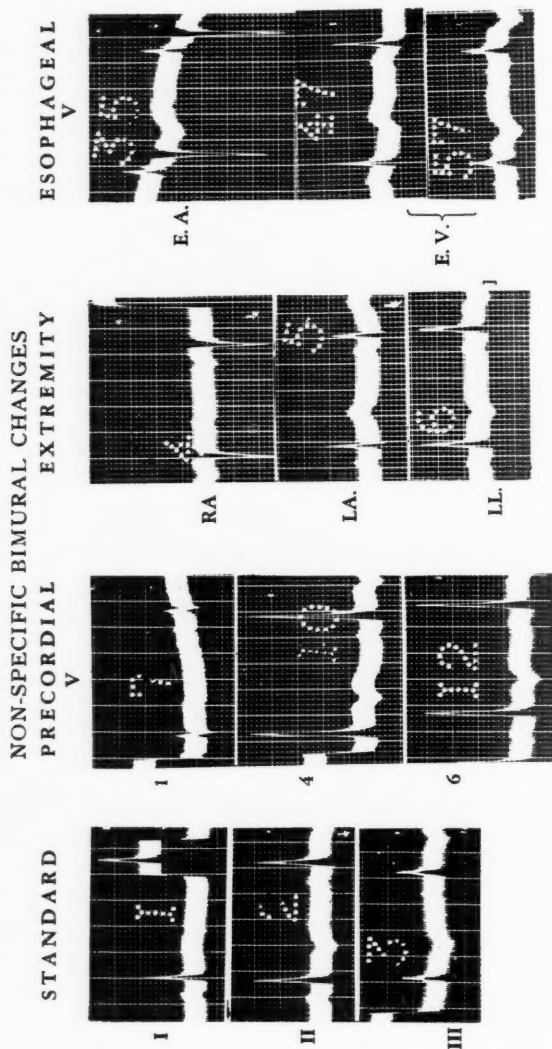


Fig. 25

Non-specific changes involving the anterior and posterior walls of the left ventricle.

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Non-Specific Bimural Changes

A. I. F., aged 47, clothing manufacturer, applied for a jumbo line concurrently with his two brothers. His family history reveals his father died at 68 of heart disease, and his mother died at 54 of diabetes and its complications. Within the past year one of the above brothers died suddenly of coronary disease at age 48 (this brother's cardiograph was of the equivocal Q_3 type and showed slurring of the initial R_2 and an absent S_1). The applicant was overweight in the past but claimed no current cardiovascular history. There was a history of rheumatism at age 10. Fluoroscopy revealed normal cardiac measurements and a transverse type of cardiac silhouette (transverse diameter 12.8). The blood pressure was 120/82/72 and the pulse rate 78. The urine was negative. A routine check by his physician revealed a constant apical murmur. The electrocardiogram (January 2, 1942) was withheld, as it was not requested previously. It revealed a low, upright T_1 of less than 1 mm., an inverted T_2 and T_3 , and a localized, unusual, M-shaped T_4 , while lead CF_2 and CF_6 were within normal limits.

Home Office examination confirmed a rheumatic history and revealed an inconstant pulmonic murmur, a constant faint, short apical murmur, and a split first mitral sound. After exercise, the apex murmur was accentuated and transmitted to the axilla. Exercise tolerance was normal. Fluoroscopy was normal and showed a small transverse heart. The special electrocardiogram in Fig. 25 shows

Abnormal standard leads with respect to T_1 , T_2 , and T_3 ;

Abnormal extremity potential LL with respect to an inverted T ;

Abnormal precordial leads V_4 , V_5 , and V_6 with respect to an inverted T ;

Abnormal esophageal ventricular leads (E. V.) at levels 47.5, 50.0, 57.5, and 60.0.

It is reasonable to conclude that progressive changes involving the left ventricle are present anteriorly and posteriorly, although the subject is asymptomatic.

Correlation and Discussion

The history of the exploratory electrocardiogram (2,4,5,7,12, 18,19,22,23,34,39,42) is well known and is most often referred to in studies of coronary occlusion. This method has, however, a wider application. Among the other important problems developed and explained by the exploratory leads are the studies of the various types of bundle branch (2,49), of hypertrophy of the right or left ventricles (16,17), of the various origins of arrhythmia (23), including extrasystoles, and of the nature of the transposition (25), of the heart.

Myocardial infarction, however, is the major topic illustrated by the foregoing cases, each of which is explained in technical terms and whether it deviates from the normal. The standard leads are correlated with exploratory electrocardiograms and what is to be probably expected pathologically. The pathological correlations are hypothetical in these cases; however, they are firmly based on the experiments and clinical materials of others (4,11,25, 26,28,32,34,35,42), who established the principles of the exploratory lead. Myers and Oren (35), have correlated the abnormal type of esophageal ventricular potentials with four cases at autopsy and found no variance with the diagnosis established by the electrocardiogram.

Quantitative evaluation is most desirable in analyzing biological and chemical phenomena. In spite of more than a third of a century of experience, accurate statistical analyses of large series of electrocardiograms are still wanting. The selection of any group of data is an arbitrary one; however, the compilations found in Table I for the voltage variations in the standard potentials, unipolar extremity potentials, and precordial leads are favored for the present. A limited treatise on this table, which is described by Wilson (9), should be consulted for details of probable errors admitted in this experience. In regard to the esophageal potentials, no adequate quantitative study of a large series of cases has yet been produced for all the deflections; however, the empirical method of analysis of form and directional changes has proved

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very valuable. The discussion of the normal subject integrates our concept of most of the empirical variations in the multiple exploratory potentials (see Fig. 2b and the text).

The P-wave needs no further discussion in relation to standard (7,8,27) and single extremity potentials. As an index of disease its usefulness in precordial ventricular leads is very dubious; it is greatly modified by varying the indifferent lead when studied in relation to a given point. Further information regarding disease or rhythm of the auricle, however, has been obtained by indirect leads over the precordial auricular region, which is a more sane procedure. With few exceptions (see Fig. 6a), particularly in auricular fibrillation (24), the character or form of the P-wave in the esophagus or stomach has been a valuable index of the location of the electrode with respect to its proximity to the ventricle, the auricle, or the region above the auricle. Because of this, esophageal electrocardiograms have become intelligible in relation to other exploratory electrocardiograms and further made it possible to expand our concept of the total electrical field of the human heart. The diagnosis of posterior myocardial infarction by the esophageal ventricular potential (7) is also made possible, although the standard leads are often doubtful, equivocal, or confused (Figs. 9a, 9b, 10b, 13a, and 13b). A modern study of exploratory leads directly on and in the auricle in animals has been reported in detail by Macleod and his collaborators (19), who describe an accession and a regression component (T-wave of the auricular electrocardiogram) of the P-wave. This recession wave is best observed in complete atrio-ventricular dissociation when esophageal auricular electrocardiograms are studied in man. The fractional equivalent of the recession wave in normal electrocardiograms is the P-Q segment following the P-wave. Larson and Skulason have recently reported the normal findings on the P-Q segment in 100 normal individuals, which is of comparative interest. No emphasis is made in the above electrocardiograms of the directional changes in the P-Q segment, which is most prominent and often modified in the juxta-auricular potentials.

TABLE II
SPECIFIC ELECTROCARDIOGRAPHIC CRITERIA OF
MYOCARDIAL INFARCTION*

Anterior Myocardial Infarction	Posterior Myocardial Infarction
(1) Q_1T_1 Criteria	(3) Q_3T_3 Criteria
Q_1 equals the only QRS_1 complex; or	Q_3 equals the only QRS_3 complex; or
Q_1 equals 1/5 or greater of R_1 ; and	Q_3 equals 1/4 or more of largest R_1 , R_2 , or R_3 ; and
$RS-T_1$ segment is transiently elevated or iso-electric; and	Q_2 equals 0.1 mv. or greater; and
$RS-T_3$ segment is transiently depressed or iso-electric; and	$RS-T_3$ segment is transiently elevated or iso-electric; and
T_1 is usually or becomes sharply inverted; and	$RS-T_1$ segment is transiently depressed or iso-electric; and
T_3 is usually or becomes sharply upright.	T_2 , T_3 are usually or become sharply inverted; and
QRS_1 is usually of low voltage; and S_2 and S_3 are often large.	T_1 is usually or becomes sharply upright.

TABLE II — (Continued)

(2) Q_4T_4 Criteria (Precordial ventricular region, particularly points 2 to 4)	(4) $Q_{EV}T_{EV}$ Criteria (Esophageal and gastric ventricular region)
Q_4 equals the only QRS_4 complex; and RS- T_4 segment is transiently elevated or iso-electric; and T_4 is usually or becomes sharply inverted or is diphasic (plus-minus) in character.	Q_{EV} equals the only QRS_{EV} complex; or Q_{EV} is $\frac{1}{4}$ or more of R_{EV} ; and RS- T_{EV} segment is transiently elevated or iso-electric; and T_{EV} is usually sharply inverted or is diphasic (plus-minus) in character.
(5) Specific criteria for multiple or combined myocardial infarction of the anterior, lateral, or posterior wall are based on the above characteristics, particularly in the series of multiple precordial and esophageal ventricular leads.	
(6) Lateral myocardial infarction is usually judged from the combined criteria in Lead I and the precordial electrocardiograms over points 5 and 6 in the anterior and mid-axillary regions, respectively, in the absence of diagnostic changes over points 2 to 4, and in the esophageal ventricular lead if taken.	

* Judged when the QRS interval is normal and there is no digitalis effect or dextrocardia present.

TABLE III
NON-SPECIFIC ELECTROCARDIOGRAPHIC
CRITERIA OF POSSIBLE MYOCARDIAL
INFARCTION

- I. In general, when the infarct is not localized by the specific criteria, there may be infarction if there is:
 1. Reduced QRS voltage in the standard leads, or
 2. Sudden development of reduced QRS voltage, or
 3. Bizarre QRS complexes in multiple standard leads, or
 4. Transient or persistent intraventricular or bundle branch block of any type other than with short PR interval, or
 5. Progressive changes in QRS-T complexes in two or more electrocardiograms, or
 6. Transient arrhythmia of any type, or
 7. A combination of some of these findings.
- II. Anterior Myocardial Infarction may be present if:
 - A. In the standard leads
 1. QRS_1 is bizarre or W-shaped; or Q_1 is $1/5$ of R_1 .
 2. Deep S_2 and S_3 appear suddenly; or S_3 appears suddenly.
 3. RS- T_1 segment is elevated and RS- T_3 is depressed.
 4. T_1 is iso-electric, inverted, or diphasic, or changing in the absence of left ventricular enlargement.
 5. A combination of the above findings is present.
 - B. In Lead IV or in the Precordial Series of Electrocardiograms (Points 1 to 6)
 1. Q is 0.4 mv. or greater; or large ($1/5$ or greater) in proportion to R—region of points 3 to 6 (Q is normally absent over points 1 and 2).
 2. R is less than 0.2 mv. at or to the left of the apical region.
 3. RS-T segment is elevated more than 0.2 mv.
 4. T is inverted or changing significantly in any region between points 2 to 6 (T-waves in this region are considered abnormal when less than 0.2 mv.).
 5. T is iso-electric or diphasic between points 3 to 6 in the absence of ventricular enlargement or toxic factors.
 6. A combination of the above findings is present.
 - C. In the extremity potentials
 1. RA—an initial R is present, RS-T is elevated, and T is iso-electric or upright.
 2. LA—an initial Q is present, RS-T is elevated, and T is inverted.
 3. LL—the R is prominent, RS-T is depressed, and T is upright.

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TABLE III — Continued

III. Posterior Myocardial Infarction may be present if:

- A. In the standard lead.
 - 1. QRS_1 , or QRS_2 and QRS_3 are bizarre or W-shaped; or Q_3 is 0.3 to 0.4 mv., or $\frac{1}{4}$ of the largest R and associated with a small Q_2 of 0.1 mv. or greater, or associated with a slurred initial R_2 .
 - 2. RS- T_3 segment is elevated and RS- T_1 segment is depressed.
 - 3. T_2 and T_3 are inverted, diphasic, or changing in the absence of right ventricular enlargement or toxic factors.
 - 4. A combination of the above findings is present.
- B. In Lead IV or Precordial Series of Electrocardiograms the
 - 1. R voltage is exaggerated.
 - 2. RS-T segment is depressed.
 - 3. T is exaggerated or changes significantly between two or more examinations.
 - 4. T is normal and there is a suggestion of the criteria in Section III A.
 - 5. T is diphasic (minus-plus) and changing rapidly.
- C. In Lead EV the
 - 1. Q is 0.4 mv. or greater; or $\frac{1}{2}$ or greater in proportion to R when the EV lead is 5 cm. or more below the auricle, as judged by the P-wave electrocardiogram.
 - 2. RS-T segment is markedly elevated electrically and has not been produced by the movement of the electrode during mechanical systole.
 - 3. T is inverted or changes significantly between two or more examinations, particularly when Lead IV is essentially normal in the region of points 4 to 6.
- D. In the extremity potentials
 - 1. LL-Q is 25% or greater than R, RS-T is elevated, and T is flat, diphasic or inverted, and associated with
 - 2. Depression of RS-T in leads RA and LA.

TABLE IV
Abridged Criteria of Borderline and Abnormal Deviations in QRS-T

	Deviation	Leads
Q or Q-S,	if deep in	I ₁₃ , II ₂₀ , III ₂₅ , LA, LL ₂₅ , IV ₂₀ , or EV ₂₀ *
R,	if the initial wave in	RA or EA
	if conspicuously large in	RA, LA, or EA or right precordium
S,	if conspicuously large in	II or LA or left precordium
RS-T,	if elevated in	I, III, RA, LL, IV, or EV
	if depressed in	I, III, RA, LL, IV, or EV
T,	if upright in	RA or EA
	if diphasic, flat, or inverted in	I, II, LL, or EV

*Numerical subscript indicates the per cent relation Q to R in standard or given exploratory lead when condition is considered equivocal or possibly significant.

This data summarizes at a glance the potential abnormalities of the QRS-T, which must be evaluated with the clinical story and progressive studies, if available.

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More recently Hecht's (34) contribution of the nature of intra-auricular and intraventricular potentials in human subjects has confirmed the experimental concept (18,19,21), of these potentials. The intra-auricular potentials are similar to indirect leads from the esophageal auricular region. The intraventricular potentials should be the mirror image of epicardial potentials over the ventricle (however, the P-wave remains electropositive in character).

The interest centered about the Q-wave (3,4,7,11,29,30,31, 32,36,37,45) as an index of cardiac disease has been justified in the last one and one-half decades. A glance at the inclusive tables listing the major changes in the electrocardiograms, whether specific (Table II) or suggestive (Table III) of myocardial infarction, confirms the significance of the Q-wave in electrocardiography. As the tables are self-explanatory with respect to the rest of the QRS-T complex, these are not further elaborated here. These should serve as a guide to and limited summary of our knowledge on this subject in relation to myocardial infarction. This evident correlation is recapitulated in graphic form (Fig. 16) for standard, precordial, and esophageal leads. A summary of borderline and equivocal deviations of QRS-T is presented for all standard and exploratory leads (Table IV) without reference to the probable significance of the deviation.

Apparent exceptions (8) to the abnormal myocardial significance of the Q-wave may sometimes arise, particularly in the esophageal electrocardiogram when the QRS interval is prolonged and the heart is rotated from its normal axis (Fig. 19a). However, occasionally myocardial infarction of the anterior and posterior walls may be diagnosed in the presence of this abnormality, as shown in some of the previous illustrations (Figs. 21 and 22). The author has shown some equivocal cases of infarction by standard leads and established the correct diagnosis from the esophageal potentials. Myers and Oren (35) have now confirmed by autopsy that these typical QT patterns in the E. V. lead are diagnostic of infarction in four cases. The reliability of the unipolar left leg lead as an aid to this diagnosis will be discussed later.

Electrocardiograms from individuals who have been therapeutically or experimentally digitalized (Fig. 23b) show changes over the entire myocardium, if we may judge from the complete exploratory electrocardiograms. The alterations of this type are largely reflected by RS-T depression in Leads I, II, LA, LL, V₄, V₆, and E. V. and U-shaped inversion of T-waves in I, II, V₅, V₆, LL, and E. V. It is known that digitalis effects a shortening of electrical systole and, therefore, of the QT interval. The PR interval, which reflects auricular and ventricular conduction, may or may not be significantly prolonged.

In the case of hypertrophy of the ventricle (48) (Fig. 14 and 15) the major changes in the electrocardiogram may occur in both the QRS complex and the T-wave of standard and exploratory leads. The frequent occurrence of right axis deviation with right ventricular hypertrophy and of left axis deviation with left ventricular hypertrophy is well known. More recently (9, 16, 16, 46), the disturbance in the form and conduction time with respect to the intrinsic interval in precordial electrocardiograms has been noted. For example, in right ventricular hypertrophy (Fig. 14) the R-wave, instead of the S-wave, becomes the chief deflection in the right precordium at points 1 and 2, and frequently the intrinsic interval, measured from the beginning of QRS to the beginning of the sharp downstroke in RS, is prolonged as compared with the normal. The electrocardiograms at these points often show, in addition to the QRS change, an inversion of T-waves in more than one position progressing to the left from the right sternal border. Although minor changes may be effected in the QRS form, the intrinsic interval in the left precordium, points 4 to 6, is usually within normal limits. On the other hand, in left ventricular hypertrophy (Fig. 15) the form of the electrocardiogram and the intrinsic interval are usually normal over the right precordium and usually considerably delayed over the left precordium. Incidentally, the T-waves in this same region (4 to 6) frequently become diphasic or inverted when left ventricular hypertrophy is present.

The esophageal electrocardiogram in the presence of hypertrophy is also of interest. In right ventricular hypertrophy (Fig. 14) the electrocardiograms in the ventricular region showed no really significant change with respect to the relative intrinsic interval, the form of the QRS, and the T-wave. This is to be expected, because the electrode is usually in the region of the left ventricle, which is supposedly unaffected. In left ventricular hypertrophy, however, the intrinsic interval is frequently prolonged and the T-wave shows significant inversion (Fig. 15). Thus, we find changes similar to those observed over the extreme left precordium. This appears logical if the electrode in each position is adjacent to the left ventricle. These changes must be differentiated from those observed in myocardial infarction, which is admittedly often a difficult problem (Fig. 16).

A simple digest of the form of the electrocardiogram in intraventricular block is difficult to formulate, as the literature on this subject is somewhat confusing. The precordial electrocardiogram in bundle branch block has been of some assistance, as shown by Wilson and his collaborators. Many cases of intraventricular block which cannot be classified by the standard leads are easily classified by the precordial series (14). In *right bundle branch block* (Fig. 17) the relative intrinsic interval is markedly delayed over the region of the right precordium, particularly points 1 and 2, where the T-waves are also frequently directed downward. In general, except for a wider QRS interval, there is a close resemblance to the effect of right ventricular hypertrophy. The left precordium apparently remains normal with respect to the relative intrinsic interval, although the form of the QRS may be slightly modified while the T-waves are usually directed up. On the other hand, in *left bundle branch block* (Figs. 18, 19a, and 19b) the right precordial electrocardiograms are usually normal with respect to the relative intrinsic interval and the T-wave. The chief deflection in these cases is usually directed downward instead of upward. Over the left precordium in left bundle branch block there is a marked delay in the relative intrinsic interval, particularly over points 5 to 6, regardless of whether it is of

the discordant or the concordant type. The T-waves in the left precordium of left bundle branch block are usually diphasic or inverted in one or both of these places.

The esophageal electrocardiogram proximal to the ventricle in right bundle branch block (Fig. 17) is not significantly altered with respect to the relative intrinsic interval and the direction of the T-waves. In left bundle branch block of the concordant pattern it has been observed (Fig. 18) that a delay in a relative intrinsic interval is present in the esophageal ventricular region and is similar to that observed in the left precordium at points 5 and 6. The T-waves in both positions are low, diphasic, or inverted. In sharp contrast to this is the esophageal electrocardiogram in left bundle branch block with a discordant pattern (Figs. 19a and 19b). It is observed in one case that there is no delay in the relative intrinsic interval and no abnormal variation in the T-wave. In another case the QRS complex is comprised of an initial, deep, slurred Q-wave consisting of almost the entire QRS deflection in this region (8). Thus, the intrinsic deflection occurs almost simultaneously with the beginning of QRS, which is similar to the right precordial, RA, and LL potentials. This leads one to believe, first, that the conduction path over the left ventricle is normal in right bundle branch block; second, that in some cases of left bundle branch block the heart may be rotated counterclockwise so that low esophageal electrodes are proximal to the right ventricle instead of the left ventricle.

The juxta-auricular potentials are probably an index of the potentials of the left ventricular cavity. In right bundle branch block (Fig. 17) the cavity (EA) shows initial and preponderant negativity of QRS. On the contrary, in concordant and discordant left bundle branch block (Figs. 18, 19a, and 19b) the cavity (EA) is electropositive during the chief QRS, and this is followed by diphasic or inverted T-waves. In right ventricular premature systoles (Fig. 20) the cavity (EA) potentials are similar to those found in left bundle branch conduction defects. These observations are in accord with the experimental observation of the cavity potentials in canine bundle branch block by Wilson and

his associates (Report to this Association, 1942). In bimural infarction with intraventricular block (Fig. 22) the cavity QRS potential (EA) is chiefly electropositive at 37.5 cm. but is bizarre at 35.0 cm. and initially electropositive at 30.0 cm. as illustrated. In anterior infarction with intraventricular block (Fig. 21) the cavity potential (EA) is principally electropositive. In these last two instances we may also be dealing with some left bundle branch conduction defect. It, therefore, appears that esophageal auricular potentials may be of limited assistance in interpreting curves with a wide QRS interval. The favored conduction from the esophageal auricular lead is probably from the left ventricular cavity, whereas the esophageal ventricular lead favors conduction from the left ventricular surface and more rarely from the right ventricular surface.

Considerable light on the form of the electrocardiogram has been obtained in the past from studies of *ventricular extrasystoles*. It has been noted that frequently the form of the electrocardiogram in Leads I and III during the abnormal ventricular cycle is often an index of the position at which this abnormal impulse arises (23). Similarly, if there is a single focus of ventricular extrasystoles, the form of the electrocardiogram on the precordium may definitely be classified as to its origin (Fig. 21 and 24). For example, when premature right ventricular extrasystoles occur (Fig. 20), the form of the electrocardiogram will resemble the form of the left bundle branch electrocardiogram on the right and left precordium. This appears to be paradoxical; however, it is easily understood if one follows the course of the impulse. It is thus expected that the relative intrinsic interval in the abnormal cycle will appear early over the right precordium and late over the left in a right ventricular extrasystole. This is illustrated clearly in a preceding figure (18). We were not fortunate enough to obtain a study originating in the left ventricle in such a series, but it is believed to be similar to the tracings seen in right bundle branch block. The esophageal electrocardiograms adjacent to the ventricle, hypothetically and practically in right ventricular premature systoles, are comparable to those obtained

over the left ventricle anteriorly; that is, the relative intrinsic interval usually appears late in the QRS cycle. The character and direction of the T-wave is unimportant in identification as to the origin of this impulse; therefore, it appears that the reason on which we base a diagnosis of right ventricular premature systole and left bundle branch block is on the form of the electrocardiogram with particular reference to the occurrence of late intrinsic deflections in the region of the left ventricle anteriorly and posteriorly. This is best determined by the path and direction that the exciting impulse takes to cover the entire myocardium.

In intraventricular block when there is a concomitant myocardial infarction, the complex form of the electrocardiogram is much more difficult to evaluate than in the absence of this pathology; however, when we carefully scrutinize the electrocardiogram and find an abnormal and consistent appearance of deep Q-waves preceding the rest of the abnormal complex, we are sometimes dealing with myocardial infarction. This is suggestively illustrated in Figs. 21 and 22. Non-specific changes may occur locally or generally, as illustrated in Fig. 24 and Fig. 25.

The contributions (7,8,12,16,25,42,43,45,46,35,36) made by unipolar extremity potentials are not to be underestimated, as too little effort has been made to correlate the facts derived from them. Besides their use in association with precordial leads to ascertain the position of the heart in the absence of roentgen assistance, the following discussion on the evaluation of deep Q_3 should be of interest to the life insurance underwriter. We should continually realize, however, that these unipolar potentials constitute the elementary potentials or the A.B.C.'s of the bipolar standard Leads I, II, and III.

*The Differentiation of Q_3 Electrocardiogram
Using Extremity and Esophageal Potentials*

It is now possible to state the complex origin of Q_3 (7,8,12,18,26,29,34,35,36,37,45) from numerous experimental and theoretical considerations; therefore, we should be able to approach its identity as physiological or pathological. There are a few basic rules and assumptions to remember. (Table V).

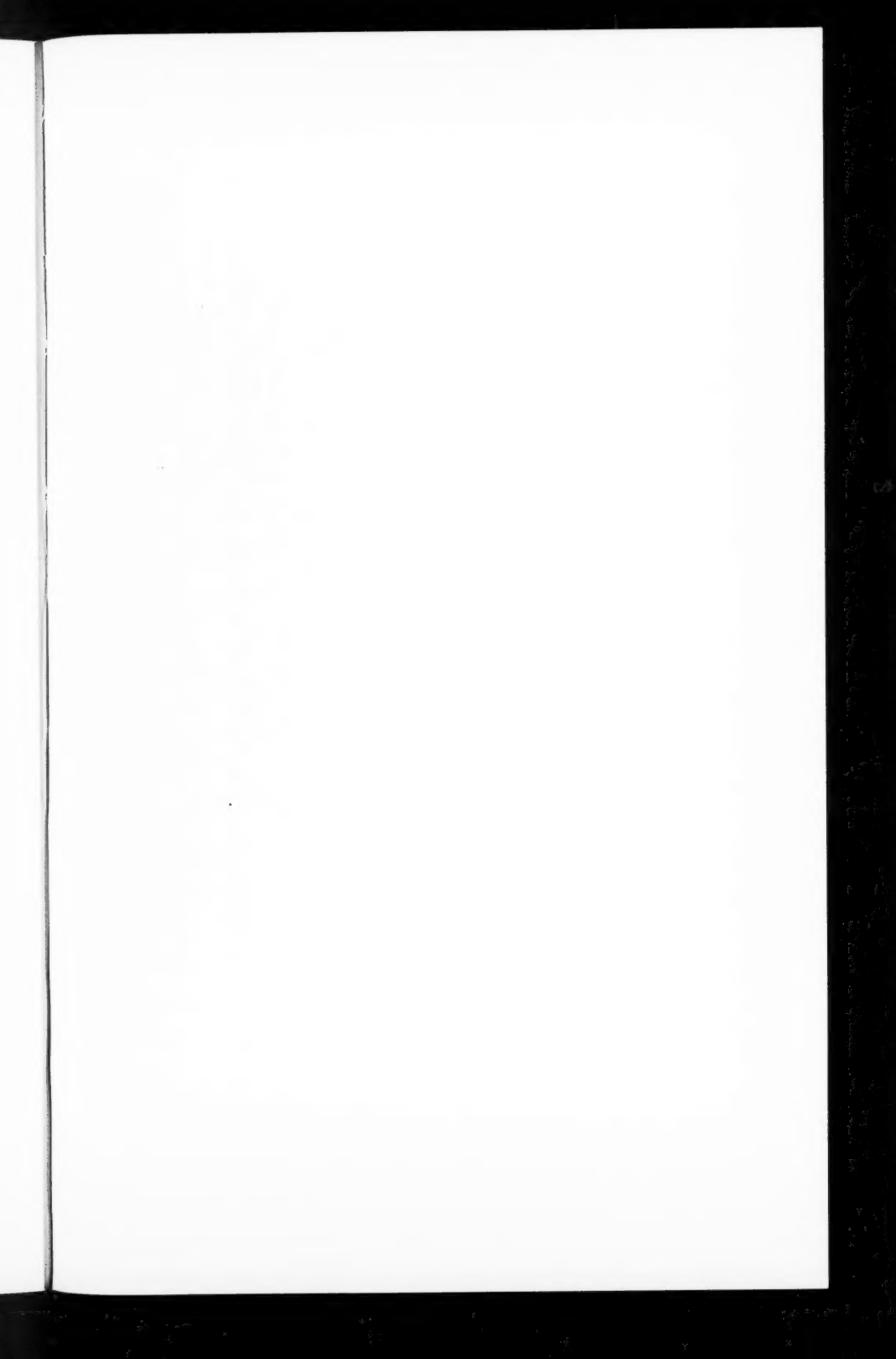


TABLE V

Basic Concept of the Electrocardiogram
for Q_3 Differentiation

- (1) The algebraic sum of the potentials at the extremities:—
$$RA + LA + LL = 0$$
- (2) Lead III is comprised of the difference in potentials:—
$$LL - LA = III$$
- (3) A positive potential (R) in LA is reflected as a negative potential (Q or S) in lead III.
- (4) A negative potential (Q) in LL is also reflected as a negative potential (Q) in lead III.
- (5) The left side of the intraventricular septum is the first portion of the ventricle to be electrically activated and therefore, it gives rise to the beginning of QRS.
 - (a) In vertical hearts—Q may be seen in LL, II or III.
 - (b) In horizontal hearts—Q may be seen in LA and I.
- (6) The potentials of the posterior and diaphragmatic surface of the heart are transmitted to the left leg (LL).
- (7) If gross infarction of this area (6) is present, it produces Q in leads EV, LL, and III.
- (8) If initial R is present in LA, it contributes to the depth of Q_3 , and
- (9) Initial R in LA produces a Q_3 when Q is absent in LL. Usually this type of origin of the Q_3 complex is physiological.
- (10) Finally, if Q in LL is greater than 25% of R in LL, we may be dealing with a posterior infarction, particularly if T in LL is inverted. This appears as an abnormal Q_3 regardless of its relative size to R in the standard leads.

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(1) The algebraic sum of the potentials at the extremities (RA, LA, and LL) due to the heart potential must equal zero at a given moment (21,44). For example, if a Q-wave is present at the left leg, this is balanced by an R potential elsewhere at the same moment to equal zero potential.

(2) Lead III is comprised of the difference in potential between LA and LL (see Fig. 26 and legend, Fig. 2a and 2b).

(3) *A positive potential (R) in LA is reflected as a negative potential (Q or S) in Lead III.* For example, if a QRS complex is iso-electric in LL at the same moment that an initial R is being inscribed in LA, then a physiological Q_3 may be present solely due to R in LA. This deflection varies considerably with respiration and may arise in horizontal types of hearts when the left ventricular epicardium faces the left shoulder or head.

(4) *A negative potential (Q) in LL is also reflected as a negative potential (Q) in Lead III.* For example, if the QRS complex is iso-electric in LA at the same moment that an initial Q is forming in LL, then Q_3 will be present solely as the result of Q in LL.

(5) Lewis (18) said that the *left side of the intraventricular septum was the first portion of the ventricle to be electrically activated and, therefore, it gave rise to the very beginning of the QRS deflection* (Fig. 1b). This is manifested as an initial Q-wave in leads facing this septal wall and, therefore, an initial R is present elsewhere. This is most easily identified as a small Q of short duration in the extreme left precordial leads of some normal subjects. In standard leads this explains why there is a high incidence of Q in Lead I in left axis deviation and a high incidence of Q in Leads II and III in right axis deviation or tendency when S_1 is present. These facts about Q are considered physiological and lead to the following correlations:

(a) The initial left septal negativity (Q) is shown in normal vertical hearts in lead LL and may be reflected as Q_2 and Q_3 of standard potentials.

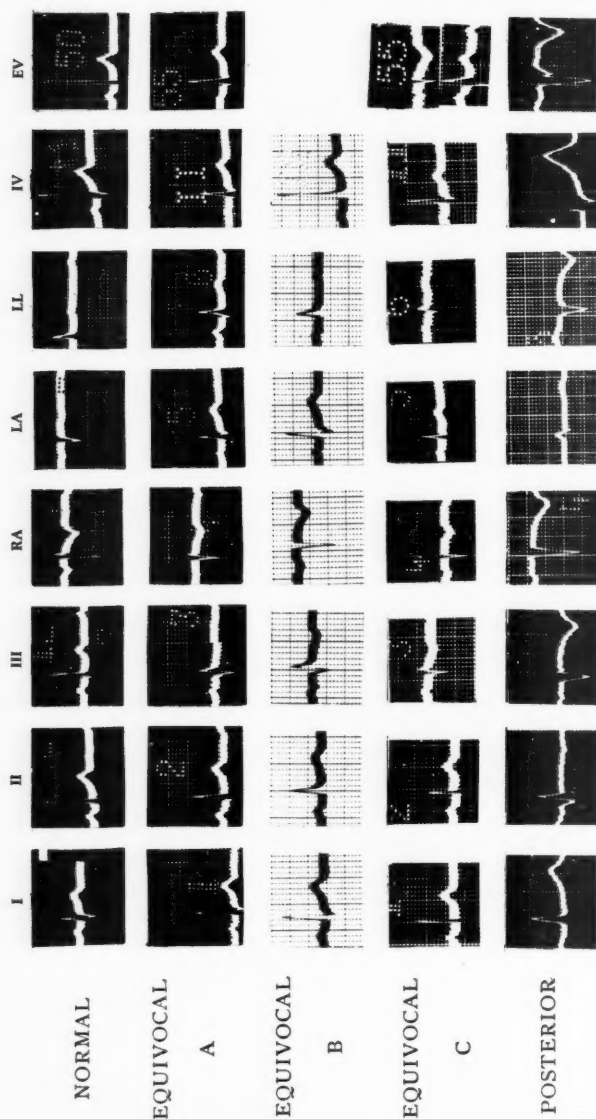


FIGURE 26
NORMAL AND ABNORMAL Q_3 ELECTROCARDIOGRAM

Fig. 26

Normal Electrocardiogram of male, aged 30. Leads I and LA show right axis deviation. Small Q_3 is probably of septal origin and is also barely identified in IV and EV. Q in LA is due to right axis shift. RA, LL, IV, and EV are normal.

A. *Equivocal standard potentials with deep Q_3 and Q_2 of 0.1 millivolt.* Female subject, aged 43, having minor G. I. complaints, not related to the heart. Initial R in LA, plus small physiological Q in LL, form the deep Q_3 . RA, IV and EV are normal. The deep Q_3 is due to a horizontal type of heart and is considered physiological.

B. *Equivocal standard potentials with deep Q_3* in male, aged 57, 5 feet, 7 inches, 177 pounds. Medical history was essentially negative. Physical examination revealed inconstant apical and pulmonic systolic murmurs, and variable blood pressures, ranging from 132/84 to 154/86. It is noted that Q_2 is absent. RA, LL and IV are essentially normal. LA shows prominent initial R due to horizontal position of the heart. This is reflected as physiological Q in Lead III, and there is only a tiny q in LL.

C. *Equivocal standard potentials with deep Q_3* in overweight female, aged 54, 177 pounds, who complained of dizziness, weakness, and a feeling of pressure in the left chest (see Fig. 13a, 13b). LL potentials are bizarre, but Q is present and proportionately large. Q_3 is largely due to a potential contribution from R in LA. The esophageal ventricular potentials at 6-month interval reveal the presence of a myocardial infarct; therefore, total reliance in Lead LL cannot be made, as it is also equivocal in this case, in which T is upright.

Posterior Myocardial Infarction (Case J. E., Fig. 5 text). Deep Q_2 and Q_3 are pathological because Q in Lead LL is abnormal. Q_3 is increased slightly by the R component from LA, probably due to a transverse position of the heart. Lead IV is normal, but Lead EV supports the diagnosis.

(b) The initial left septal negativity in normal horizontal hearts is physiologically present as a small Q in the LA potential.

This, in turn, may be reflected in Lead I as Q_1 (as all the negative potentials of LA are deflected downward in Lead I if they are not balanced out by potentials in RA).

(6) *The potentials of the posterior and diaphragmatic surfaces of the heart are transmitted to the left leg.* The chief QRS potential in normals is electropositive, but this may be preceded physiologically by a small Q resulting from left septal negativity (5a). The forms of the normal left leg potential and esophageal ventricular potentials are very similar and described above (Fig. 2a and 2b).

(7) *When gross infarction of the posterior and diaphragmatic wall of the heart exists, a deep Q -wave of the intraventricular type replaces the normal QR potential of the esophageal ventricular lead (E. V.). This abnormal, deep Q -wave is usually transmitted to the left leg (LL). Finally, this abnormal Q in LL is reflected into Q_3 pathologically with or without a contribution from the LA potential (R) as described above (3) (Fig. 26 and legend).*

(8) It is noted from cases above and others in the literature that when Q is present in LL due to infarction posteriorly, R is often the initial deflection in the LA complex, and, therefore, Q_3 is often very deep. The R in LA may be compensatory to the formation of the posterior infarct. The heart that is infarcted may change its spatial relationship and accentuate this negative reaction (Q) in Lead III.

(9) Further, if there is no Q in the LL potential and an initial R is present in LA, we are primarily dealing with a *positional* or *physiological* Q_3 , although hypertrophy may have produced the peculiar anatomic relation. Therefore, unipolar extremity potentials do appear to be of assistance in our Q_3 problem.

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From Table II, it is noted that no specific criteria have been elaborated for myocardial infarction in relation to extremity potentials. This is correct, but the assistance they may give when precordial leads fail and when esophageal leads are not available may be indispensable if we earnestly endeavor to differentiate Q_3 (36).

(10) It is reasonable to conclude from observations to date that if Q in LL is *proportionately large or greater than 25% of R in LL , we are dealing with a posterior type of infarction. This is particularly true if Q is broad, 0.04 of a second or greater, and the QRS complex is followed by an inverted T -wave in LL . This rule has exceptions, and in this paper were demonstrated in Fig. 9b, in which Q_3 was absent in the presence of a posterior infarct identified by esophageal potentials.*

Summary: The application of standard, unipolar extremity, precordial, and esophageal leads in electrocardiography has resulted in a rational and graphic concept of the entire electrical field about the normal and diseased heart.

The precordial potentials are best used to establish the presence of anterior myocardial infarction and to differentiate the various types of intraventricular block and ventricular arrhythmias.

The esophageal potentials are best used academically to illustrate pathology of the left auricle and adjacent ventricle. They have served to establish the presence of posterior myocardial infarction when all other leads were equivocal. Autopsy confirmation of pathology disclosed by the esophageal lead has been established by Myers and Oren (35).

The esophageal auricular potentials may represent potentials from the left ventricular cavity and may be of limited assistance in evaluating curves showing a wide QRS interval.

The extremity potentials serve to explain the patterns of the standard leads, of which they constitute elementary potentials.

The differentiation of physiological and pathological Q_s , using extremity and esophageal potentials, has been discussed.

A table of standard, extremity, and precordial potential values is reprinted.

Tables of Specific and Non-Specific Criteria of Myocardial Infarction are presented.

A tabular summary of borderline and abnormal characteristics of QRS-T is presented for standard, extremity, precordial, and esophageal potentials.

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PRESIDENT STREIGHT—Dr. Lauritz S. Ylvisaker, Medical Director of the Fidelity Mutual Life Insurance Company, will now present a written discussion of Dr. Nyboer's paper. Dr. Ylvisaker!

DR. YLVISAKER—Electrocardiography has now been extensively used in life insurance underwriting for approximately fifteen years. It is generally accepted that we shall want to continue the use of this procedure because it is the most valuable practical method of securing objective evidence of the condition of the myocardium in health and disease. In view of the fact that improved health conditions allow more and more individuals to reach later life, our exposure to death, disability and impairment from myocardial involvement due to coronary sclerosis will become greater than ever before. It is likely, therefore, that we shall, in the future, rather want to increase the use of electrocardiography in order to prevent undue mortality losses in the upper age group.

In reviewing Dr. Nyboer's excellent paper, one is immediately impressed by the remarkable progress which has been made. When we were first called upon in the early thirties to apply the information from electrocardiograms, we had only the information which had been accumulated from the use of the three standard leads.

This information was helpful, but relatively little was known regarding the basic fundamentals of electrocardiography as we know them today. As a result, we were unable to analyze findings as they appeared and were forced, in our consideration of presumably healthy applicants for insurance, to apply information regarding electrocardiographic findings which had empirically become identified with heart disease. We soon learned that some of these findings which previously had been thought due to disease might occur in both normal and abnormal hearts. As we were unable to make this differentiation, we had to be unduly conservative in our approach.

The problems which arose stimulated study in our own groups for underwriting purposes and added stimulus to research in electrocardiography otherwise. As a result, much information has been accumulated and Dr. Wilson reported on this progress to us in his very valuable contributions in 1937 and 1942. He gave us a summary of the fundamentals on which future electrocardiographic thinking will probably be based, described the electrical activities as they occur in heart muscle in health and disease and how they give rise to the various deflections in the electrocardiogram. He showed us how to analyze the three standard leads by breaking them down into their component parts by means of the unipolar extremity leads. He emphasized the importance of multiple precordial leads and the need of studying electrocardiographic patterns even more than the individual deflections. He described the main electrocardiographic patterns and showed us how to recognize them on the basis of the findings in the standard, precordial and unipolar extremity leads.

The contribution of Dr. Nyboer, who is a student of Dr. Wilson and who has become a student of electrocardiography in his own right, is a most welcome addition to the previous contributions of Dr. Wilson. Dr. Nyboer has given us the most complete and clear picture of electrocardiographic patterns as they are now known and has contributed also the information provided by the esophageal leads to further complete the picture.

Few of us perhaps have ever made an esophageal electrocardiographic study and few of us will ever make one on an insurance applicant. His clear picture of these patterns, however, with the addition of the information provided by the esophageal leads will always serve as a helpful background on which we can better analyze less complete studies as they may come to us. It would seem impossible for any of us to add in any way to his study unless a diagrammatic approach such as is shown in the following figures will help us to visualize these patterns more clearly.

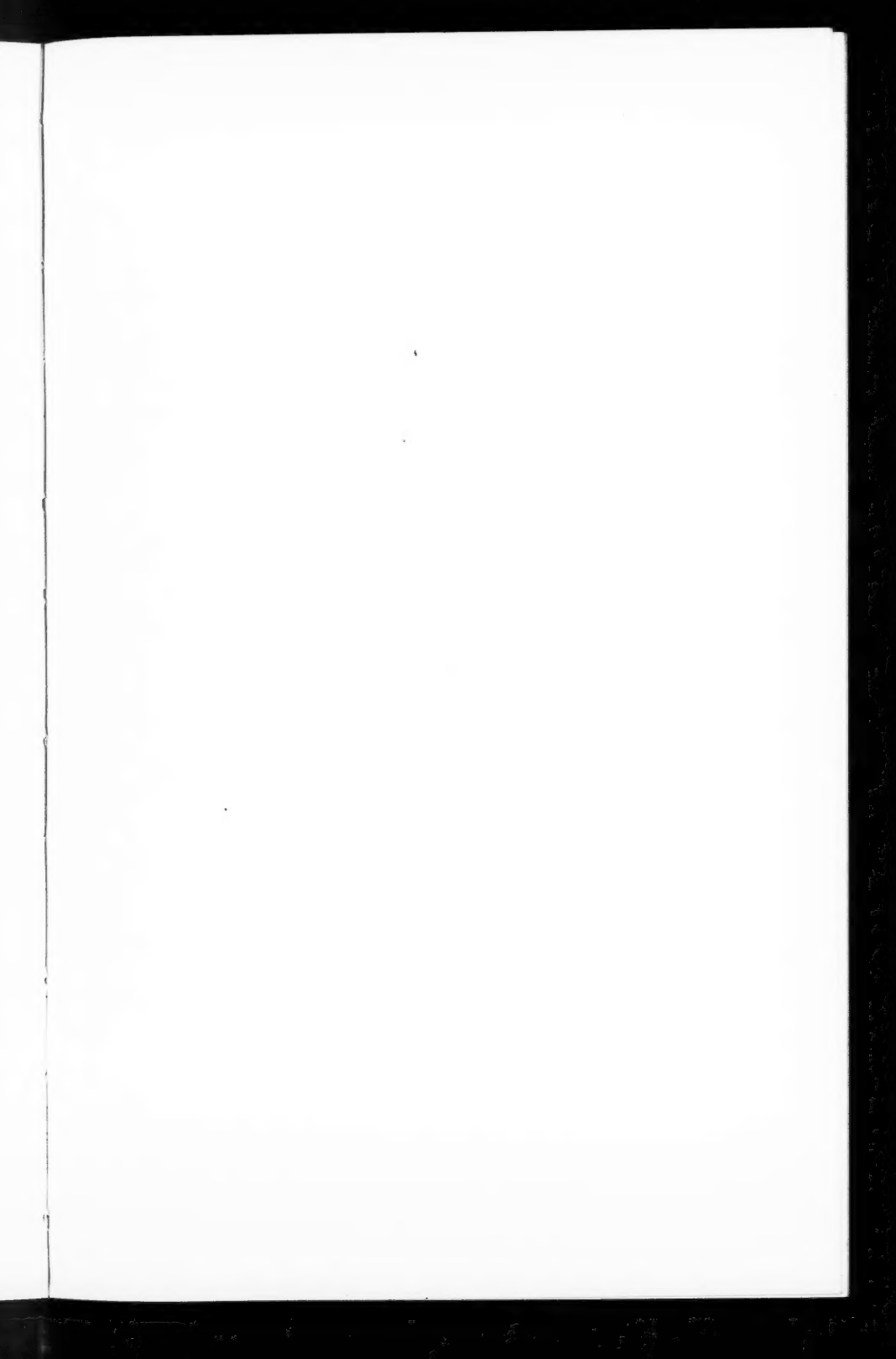
Figure I gives us a picture of Left Bundle Branch Block and shows particularly the relation of findings in the precordial leads to the actual lesion in the left branch with resulting delay in the appearance of the intrinsic deflection on the left side and no delay on the right side.

Figure II gives us a picture of Right Bundle Branch Block with delay in the appearance of the intrinsic deflection on the right and no delay on the left. Similar diagrams of other electrocardiographic patterns are available and may be helpful in clarifying the picture of these patterns.

It is not enough, however, that we learn to understand clearly the electrocardiographic patterns of advanced myocardial disease. We seldom, in fact, see these patterns in applicants for life insurance. We meet much more frequently with borderline patterns, which may or may not represent disease and which frequently lead to erroneous conclusions if adequate studies are not made. A thorough analysis of these borderline patterns on the basis of the methods suggested by Dr. Wilson and Dr. Nyboer for the purpose of drawing clearer lines between the normal and abnormal is most urgently needed and we hope such an analysis can be presented at one of our future meetings. We can all contribute to the understanding of these borderline patterns by preparing records of cases which come up for our consideration with adequate studies whenever possible.

Figure III shows the electrocardiogram which we make routinely in order to be prepared to analyze any unusual finding which may appear. We have included the three standard leads, the third lead on deep held inspiration, six precordial leads, V 1-6, a single ensiform lead and the three unipolar extremity leads.

Figure IV. At times the problem at hand may call for additional precordial leads either to the right or left of the usual V 1-6 records.



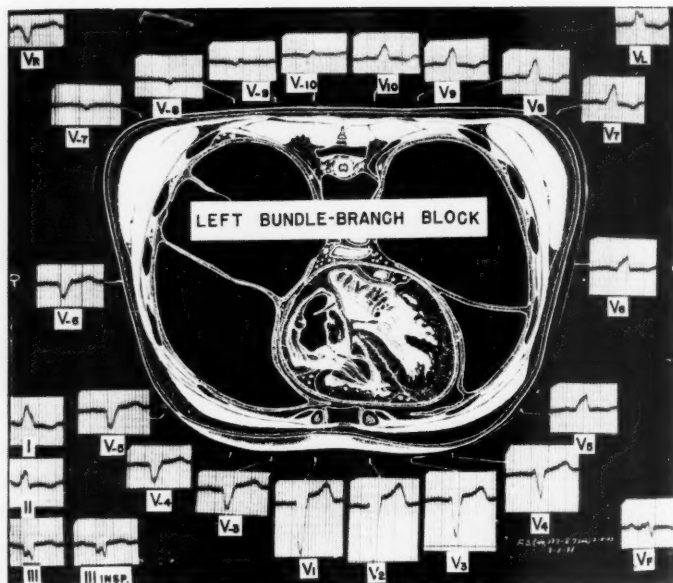


FIGURE 1

Cross section of the body at the level of the heart showing the intraventricular septum, right and left ventricle and the relation of the findings in the precordial leads to these structures in Left Bundle Branch Block. The delay in the appearance of the intrinsic deflection characteristics of this pattern is shown in the V_5 to V_{10} leads. The standard leads are shown at the lower left, the unipolar extremity leads, V_R and V_F , in their respective relation to their points of origin, right arm, left arm, and left leg.

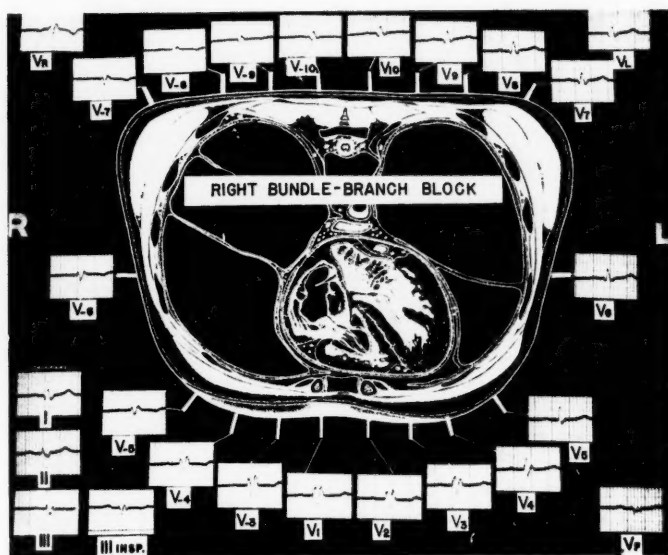


FIGURE II

In Right Bundle Branch Block, the delay in the appearance of the intrinsic deflection is shown over the right precordium from the lead in the right mid axilla V_6 to and including V_3 with no delay in the V_4 to V_6 leads over the left precordium

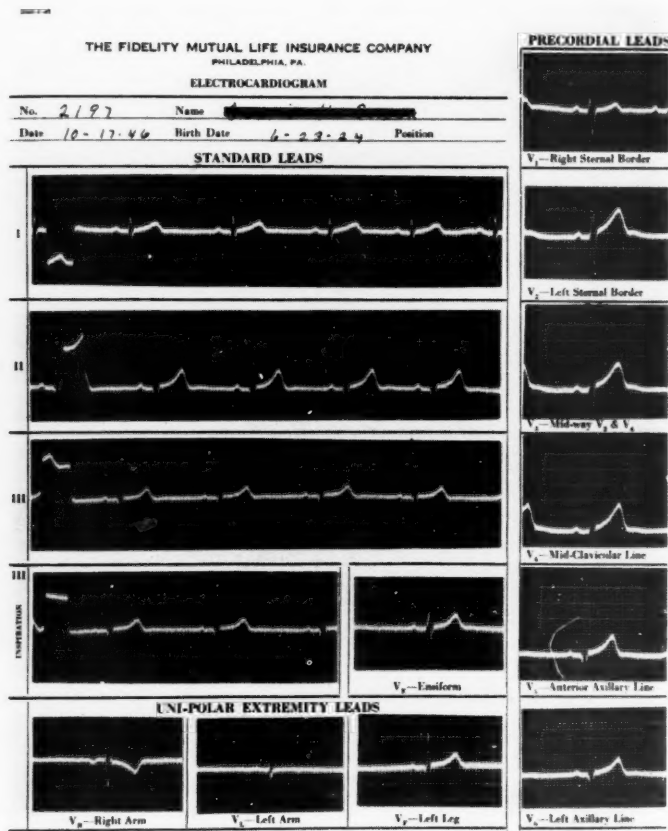


FIGURE III

Routine Electrocardiogram. The precordial leads V_1 to V_6 , the ensimform V_E , the unipolar extremity leads, V_R , V_L , V_F , and Lead III in deep inspiration all add to the information which may be needed in the interpretation of the findings in the three standard leads.

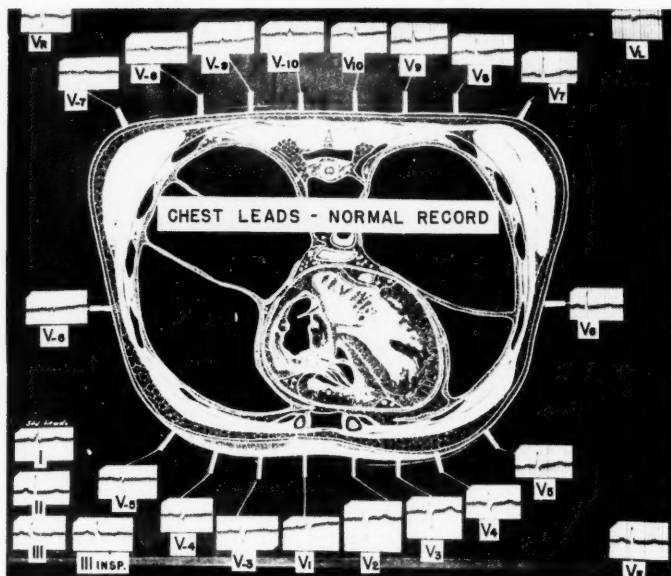


FIGURE IV

Normal Record for comparison with Figure I and II and other abnormal patterns.

CHEST LEADS AT VARIOUS LEVELS

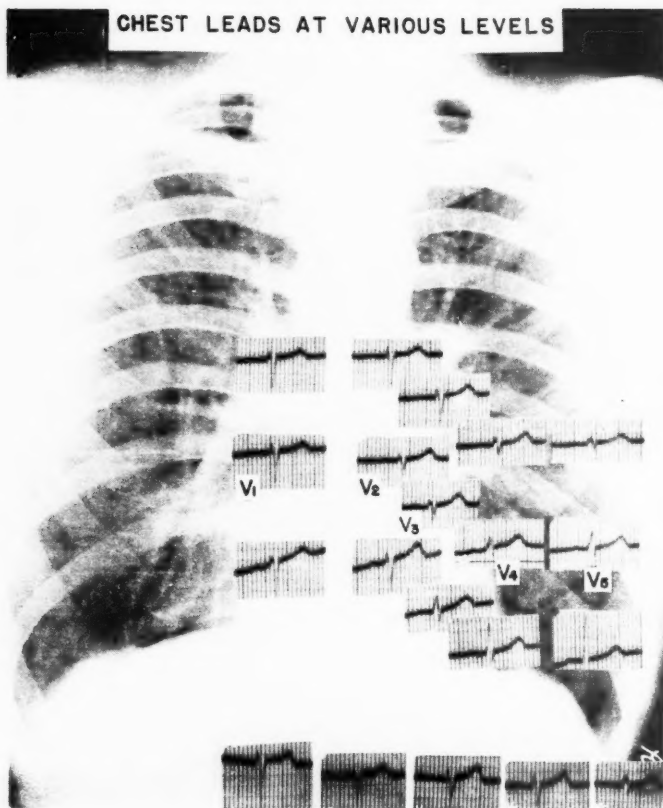


FIGURE V

Precordial leads at three different levels, one interspace above and one below the usual V_1 to V_5 positions which may be helpful in the interpretation of bizarre patterns occasionally found in the usual V_1 to V_5 positions. The three level records shown were made to study the unusual QRS complexes found in the original record in lower part of Figure. The questionable findings in the V_2 to V_5 positions are not present in the records at the lower level and are probably due to the relation of the electrode to the position of the heart and therefore not significant.

Figure V. At other times it may be necessary to make precordial leads at several chest levels to rule out complete or incomplete right bundle branch block on the right side and to rule out high lateral infarction on the left side of the precordium.

With such studies on records with borderline patterns, we should accumulate the information which will help us to develop an even more "rational and graphic concept of the entire electrical field about the normal and diseased heart" (Nyboer). This will make electrocardiography an even more practical and reliable aid in underwriting and help to prevent undue mortality losses without excluding those with no evidence of heart disease from the benefits of insurance.

PRESIDENT STREIGHT—Our next speaker is Dr. Charles H. Best, Professor of Physiology at the University of Toronto Faculty of Medicine. He will read his paper on "Thrombosis from the Physiological Viewpoint." Dr. Best!

THROMBOSIS FROM THE PHYSIOLOGICAL VIEWPOINT

By C. H. BEST, C. B. E., M. D., F. R. S.

*Department of Physiology and Banting and Best Department of
Medical Research, University of Toronto.*

The period between 1860 and 1915 might be described as the "pathological era" in the study of thrombosis. Virchow, Zahn, Eberth and Schimmelbusch, W. H. Welch, Zurhelle, Osler, Bizzozero, and many others, wrote their classical papers on the pathological findings in clinical and experimental thrombosis. It was established that thrombosis of blood vessels might be produced by the application of caustics or by mechanical injury to the intimal surfaces of blood vessels. These procedures resulted in the accumulation of blood platelets on the wall of the vessel at the point of injury. Leukocytes then collected at the margin of and between the masses of platelets. Fibrin was subsequently laid down in large amounts. The growth of a thrombus down stream was established by histological studies.

The structure of white thrombi has interested students since the work of Virchow in 1863, who believed that the whitish masses which are frequently observed in blood vessels under certain conditions, were formed by white blood cells, disintegrated fibrin and red blood corpuscles which had lost their haemoglobin. It was Mantegazza in 1869 who demonstrated the fact that the white thrombus does not arise by a gradual change from a red thrombus but originates in a form very similar to that in which it finally appears. Mantegazza thought the thrombus was formed from white blood corpuscles and fibrin and many of the subsequent workers had the same conception of the mode of origin. A signal advance was made in this field in 1882 by Bizzozero who watched the formation of a white thrombus under the microscope.

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He found that when he caused slight damage in a surface of an arterial wall of a small mammal, a thrombus was soon produced. A whitish mass was formed almost entirely by blood platelets. There were a few white blood cells.

In 1929 investigations on thrombus formation were initiated in Toronto and we (Best, Cowan and MacLean) obtained direct evidence in confirmation of the work of Bizzozzeri, that platelets were deposited on a distal or down-stream side of the primary mass. We used a glass cell. The proximal or up-stream side of a mass of platelets changes relatively little. Definite growth can be noted on the right or distal side. This phenomenon is shown even more clearly when growth of the thrombi on the scratch is watched. Observations made upon the formation of thrombi on the scratch and also in the narrow parts of the cannula, would appear to show that this process may take place without marked slowing of the bloodstream. On the other hand it is certainly true that one of the causes of thrombus formation is slowing of the stream and, as a matter of fact, in the peripheral parts of the glass cell where the blood flow is slowed, you can see the deposition of platelets on many occasions before they appear on the scratch. When there is a rough surface such as a scratch on the glass tubing, thrombi form quickly in spite of the fact that the blood stream is moving very rapidly. It may be that the scratch forms eddy currents which we can not see but we are able to detect none before the clumps of platelets are clearly visible. The foreign surface is obviously an important factor in this rapid formation of thrombi. (A *Moving Picture Film* illustrating this formation of thrombi was then shown.)

After deposition of platelets in the glass tube or in the injured femoral vein or coronary artery, the blood flow is slowed and clotting may occur. Under these circumstances one gets either white thrombi or mixed thrombi or a clot, i.e., the red type of thrombus. (6 SLIDES illustrating the production of thrombi in various situations were then shown.)

The body's defenses against excessive deposition of platelets and the formation of a thrombus are multiple. The maintenance of a smooth intimal lining of the blood vessel in which the platelets may play an important part, the control of the number of cells of the blood and the regulation of the amount of those potent agents which augment or inhibit the clotting system or the formation of thrombi are all important factors.

The "physiological era of thrombosis" may be said to begin with the isolation of heparin and I would focus your attention particularly this afternoon on this anti-coagulant. While a search of the earlier literature reveals the fact that several investigators realized that certain mamallian tissues contained one or more anti-coagulants, an active fraction was first isolated in Howell's laboratory in 1916 by McLean. This was subsequently investigated by Howell and Holt in 1918. Howell named the substance "heparin" (hepar - the liver) and he and his colleagues have studied its distribution, chemical properties and physiological significance. For some time heparin was prepared exclusively from the liver tissue of the dog. It was very difficult to secure sufficient amounts of a product completely satisfactory for physiological experiment. The clinical application had not been developed.

In 1928 I initiated a project in Toronto which had as its objective preparation of a more potent and more highly purified heparin, and a study of the effect of this substance in preventing thrombosis. We found that it was possible to prepare active fractions from ox liver and subsequently Charles and Scott in 1933, showed that this substance was present in varying amounts in a great many tissues of the ox. Lung, liver and skeletal muscle giving in this order the best yield. The clinical and physiological studies on heparin were developed in Toronto with the collaboration of Dr. Gordon Murray and Dr. L. B. Jaques, and in Stockholm by Jorpes and his colleagues.

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Chemistry of Heparin

I have no intention of discussing this subject in detail to-day. It will suffice to state that heparin is a mucoitin polysulphuric acid. It can be prepared as either the barium or sodium salt. These crystalline salts are of constant chemical composition and physiological potency. Attempts to make heparin synthetically have thus far failed. There is an International Standard of heparin which contains 100 units per mg.

The Action of Heparin on the Clotting System of Blood

The most significant property of the anti-coagulant heparin is its marked ability to delay the clotting time of blood. Since prothrombin is present unchanged in heparinized blood the important anti-coagulant action of heparin is presumably due to the effect on the first stage of clotting. Heparin also acts as an anti-thrombin but only in the presence of a co-factor present in plasma. At present there is great interest in the anti-tryptic action of heparin. This substance inhibits both crystalline trypsin and plasma trypsin. Heparin possesses a very striking property of forming compounds with protein and other complex bases. The reaction of heparin with protamine is of great interest and as first shown by Chargaff and Olson, it has a greater affinity for protamine than for the plasma proteins. Thus it is possible to use protamine for the determination of heparin in blood or to neutralize the heparin which has been added to blood. Both of these procedures may have important clinical applications.

It has been found that when a system similar to that used for the determination of prothrombin time, is used for the assay of heparin, certain of the synthetic polysaccharids are more potent than heparin. On the other hand their anti-coagulant activity measured on fresh whole blood, is only a fraction of that of heparin. The lengthened coagulation time of blood after a single intravenous injection of heparin, passes off quickly. Heparin is lost from the blood through

excretion from the kidneys and also by inactivation. The latter process is presumably caused by the enzyme "heparinase" which was discovered in our laboratory by Jaques.

Single large doses of heparin given intravenously are wasteful and for this reason either continuous intravenous administration has been used clinically, or more recently some workers advocate the subcutaneous administration of the material in various types of menstruum.

Liberation of Heparin in the Body

Holmgren and Wilander showed that the mast cells contain large amounts of heparin. These cells, found chiefly in vessel walls and in tissues where marked disturbances in the blood are taking place, i.e., in liver, lung, intestines, and so on, suggest that the physiological role of heparin in the body may be to prevent intravascular clotting of the blood which might be initiated by various physiological processes. Jaques and Waters reported the isolation of heparin from the blood of dogs in anaphylactic shock and Wilander from the blood of animals in peptone shock. In the isolated liver Rocha e Silva found that when peptone was added to Tyrode solution, perfused through liver, no heparin or histamine were released. When citrated or defibrinated blood was added to the peptone a little heparin and histamine were liberated. When peptone was added to blood preserved in silicone plastic (Jaques, Fidler, Feldsted and Macdonald) and this blood which contained no anti-coagulant perfused through the liver, Rocha e Silva, Jaques and Scroggie have recently found that large amounts of heparin and histamine are liberated. If heparin is added to the peptone-containing blood, no heparin is released from the liver. It would appear, therefore, as the above mentioned authors have suggested, that the changes in blood which heparin may normally prevent may be those which cause its secretion from the liver into the blood.

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Heparin and Thrombosis

It has been shown experimentally in a great variety of ways that heparin prevents the formation of thrombi in experimental animals. The glass tube which you have seen can be kept clear of thrombi in most species that have been studied. Heparin prevents the experimentally produced coronary or intraventricular thrombi. There is a latent period after the injection of heparin before the effect of thrombus formation is apparent. Under certain experimental conditions this may be from 15 to 50 minutes. The interval under clinical conditions is not known. The mechanism by which this anti-thrombotic effect is produced is not yet completely clear. The action may be the same as that which prevents the coagulation of blood or there may be some other process causing agglutination of platelets which heparin inhibits. This is still an unsolved and a very important problem.

Clinical Problems

If I were able to discuss the clinical as well as the physiological aspects of thrombosis, all the literature on the use of both heparin and dicoumarin would have to be reviewed. There is little doubt that either of these agents may, under favourable conditions, prevent thrombus formation in human subjects. The control of clinical studies always present much greater difficulty than is encountered experimentally.

Heparin must be given intravenously or subcutaneously. The latter procedure is not fully established clinically. The action of heparin given intravenously begins after a relatively short latent period and disappears rapidly after the administration is terminated. Heparin may be very rapidly neutralized by the administration of protamine. This clinical use of protamine for this purpose is by no means fully established. A great deal more work must be done before it can be safely recommended.

Dicoumarol

The story of dicoumarol begins with the work of Schofield in Canada and Roderick in the U. S. A. The origin of a haemorrhagic disease in cattle was traced to spoiled sweet clover hay. Dr. Link has reviewed the brilliant work of his group on the isolation, crystallization and synthesis of the anti-coagulant and its chemical and pharmacological properties. Dicoumarol acts quite differently from heparin. The former has no effect on blood *in vitro* but causes a lowering of blood prothrombin through a pharmacological effect on the liver. Dicoumarol is administered orally, it has a relatively long latent period (48 hours) before it becomes effective and its action persists for days after cessation of administration. Its effect can be neutralized in part by vitamin K or by a blood transfusion. Dicoumarol-like heparin prevents experimental thrombosis and favorable clinical results have also been reported.

The extent to which the coaguability of blood must be lowered by heparin or dicoumarol to prevent thrombus formation, is very difficult to determine in human patients. There is probably great individual variation in relatively healthy people and even more as a result of disease. It is understandable that very few attempts have been made by clinicians accurately to determine the minimum amounts of heparin or dicoumarol which are required. There is a great need for this data. These useful tools have sharp edges and overdosage is dangerous. On the other hand it would be all too easy in a study of the incidence of thrombosis, for a clinician to report favorable results with a dosage which had no real effect.

The relative clinical merits of heparin and dicoumarol can only be decided by very carefully controlled clinical studies. As some workers have suggested the agents may be used together to secure both an immediate and a prolonged effect.

It is very dangerous for a physiologist, even if he has a medical training, to make this excursion into clinical problems. I believe, however, that many clinical experts in this field realize the need

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of further careful study of both heparin and dicoumarol. Experimentalists and clinicians alike will agree that the solution of the fundamental problem — the detection of those patients in whom thrombosis is likely to occur — would represent a very great advance and would facilitate the accurate evaluation of anti-thrombotic agents.

PRESIDENT STREIGHT — Thank you, Dr. Best, for your very fine paper. I am particularly interested in the topic Dr. Best was discussing because I, unfortunately, had a serious attack of pulmonary emboli a couple of years ago, and I was treated with heparin, I think successfully, otherwise I would not be here, so I am very much interested in that topic.

The topic is now open for general discussion, and I am sure that Dr. Best would be glad to answer any questions that any of you may care to ask him.

DR. RICHARD S. GUBNER — I would like to ask whether Dr. Best believes that heparin plays a physiological role?

DR. BEST — I don't think it is finally established that heparin has a physiological role. The evidence, as Dr. Gubner probably knows, is that it occurs in the body as apparently a specific enzyme. In shock, you get huge amounts liberated. I think the evidence is still circumstantial that you find it in amounts, where it would be of use if you liberated it. I don't know what to call heparin. It isn't a hormone. A hormone is a material that is made in one gland and carried somewhere else to be utilized. Heparin, if you are going to classify it in that category, is very generally distributed wherever the mast cells are. It acts on the blood vessels presumably just where it is liberated. It has a physiological significance. My intuition tells me it is physiological. We find it there in amounts that vary under different conditions, but more physiological work is needed to prove that it functions under normal conditions.

DR. CECIL C. BIRCHARD — Mr. Chairman, I would like to ask Dr. Best through you, sir, if he can give us his ideas of

the actual sequence of events in coronary thrombosis, from the first lesion, whatever it may be, on through post mortem?

DR. BEST — Well, Dr. Birchard, I am dodging that one. The only type of coronary thrombosis I know is the one we produce in dogs, and there, of course, it is under control. You just make a little scratch or irritate the intimal surface of the coronary artery, and the sequence is that there the deposition of the platelet is the first thing you see. Dr. Paterson, who was with Dr. Falconer and Dr. Gray and Dr. Noble and myself of the Class of '25 in Toronto, could give you a better answer than anybody I know of. He worked at the inception of thrombosis in human material, and he finds this little lesion in the intima, and the blood getting back of that intima. I musn't break my rule about talking on clinical material.

PRESIDENT STREIGHT — Did you want to say something?

DR. E. S. DILLON — I was about to ask Dr. Best about his experiences in the treatment of clinical coronary thrombosis with heparin, whether it is useful in preventing it, but he has already answered that.

DR. BEST — No, I think one should emphasize that experimental results produced in clinical coronary thrombosis bears no relation whatever to what we see pathologically or in the clinic. It is just a demonstration that you can produce and prevent it. It doesn't lead us to the use of heparin at all, without clinical studies and I think clinical studies on that particular type of thrombosis are pretty hopeless. I mean, you have no warning.

PRESIDENT STREIGHT — Thank you, Dr. Best, for your splendid paper. Our next speaker holds an eminent position in the field of gastro-enterology being the author of textbooks and numerous articles on the subject. He is Consulting Gastro-enterologist at Mount Sinai Hospital and Associate Editor of the "American Journal of Digestive Disease and Nutrition". He will deliver his paper on "Digestive Ulcers, Their Significance and Prognosis." — Dr. Crohn!

DIGESTIVE ULCERS, THEIR SIGNIFICANCE AND PROGNOSIS

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In recent years new facts and more modern viewpoints have entered into the problems of peptic ulcer, views that alter our fundamental clinical concept of the disease, and concepts that modify the estimates of insurability of the affected subjects. The acceptable criteria regarding peptic ulcer are anything but static. Much new statistical data, a more modern psychosomatic approach, the effects of the recent world upheaval, a reconsideration of many of the methods of medical and many, too, of the surgical plans for reconstruction, these all deserve consideration if we are to maintain an ultra-modern concept of the problems. The modernization of the entire schema by altering fundamental concepts influences some of the questions regarding insurability; among these factors are increasing incidence of ulcer, more successful medical management, reduction of the periods of disability, and perfected methods of surgical approach with rapidly vanishing mortality tables.

Increasing Frequency

It is a question whether peptic ulcer is occurring with greater frequency in our population, or whether the apparent increase is due to our accentuated vigilance and acumen in recognizing the characteristic lesions. The improved efficiency in roentgenographic identification will alone explain the apparent gain in number of cases; the better clinical teaching and awareness is still another factor; the widely heralded statistics from Army and Navy sources constitute another force that makes the problem of peptic ulcer seem to be one of increasing incidence and importance. In 1929 Hurst and Stewart (1) from the Leeds General Infirmary in England published their findings in 4,000 consecu-

tive autopsies. Chronic ulcer or its scar was found in the duodenum in 5.75 per cent of bodies as compared with 4.33 per cent in the stomach. Robertson and Hargis (2) noted healed or active ulcers in 11.85 per cent of cadavers at the Mayo Clinic. Jennison (3) of the Metropolitan Life Insurance Company found an incidence of 7 per cent among its employees.

In a recent survey of clinical material at the Peter Bent Brigham Hospital in Boston, E. S. Emery, Jr. (4) published some interesting figures. The specialty of Gastroenterology covers a very wide field in clinical medicine. Approximately 17 per cent of outdoor cases, 23 per cent of the ward cases and 16 per cent of the private cases were seen because of a gastrointestinal disorder. Approximately 60 per cent were functional, 40 per cent organic. Between 18 per cent (private practice) and 35.8 per cent (out-patient department) of these organic diseases consisted of peptic ulcer cases. In the X-ray Department of the hospital alone 35.6 per cent of the organic diagnoses were those of peptic ulcer, well over one-third.

All classes of persons, all industries and professions are herein represented. It was at one time thought that ulcer affected to a greater extent the intelligentsia and particularly the professional classes. But this too is contradicted by the statistics of Hinton (5) who showed as great an incidence in draymen and manual laborers as in the white collar class.

Males, predominate greatly over females, due to the far greater incidence of duodenal to gastric ulcer; while gastric ulcer may affect male and female in approximate equality, duodenal ulcer is generally ten times more common in men than in women. If the "ulcer constitution" as depicted and described by Draper, Franz Alexander, Moschcowitz and others be accurate, then we can understand why men and duodenal ulcers predominate. They have the greater economic drive, assume far greater responsibility, assemble greater ambitions, constitute the front line in the industrial competitive strife, secrete more gastric juice of higher acidity titer, have worse habits of irregular meals, abuse smoking and alcoholic drinking, and as patients with an established

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diagnosis are more likely to be antagonistic to treatment, more recalcitrant, less well-disciplined in the matter of dietary care and personal hygienic restrictions.

All collected statistics from the Military Branches of the Government Services emphasize the importance of the ulcer problem and the great incidence of ulcer cases in its personnel. While in civilian life 35.8 per cent of the gastrointestinal cases were ulcer, in the British Army 55 per cent (Tidy) (6), in the German Army 33 per cent, in the American Army 41 per cent were peptic ulcers, of which 88 to 95 per cent had digestive symptoms prior to their entry into military service. The Armed conflict intensified a civilian complaint; the physical hazard, the emotional and mental strain, the dietary rigidity emphasized and developed the taint emanating from urban and rural sources. The Great Plague of the present war was not dysentery or cholera or diphtheria or influenza epidemics as in former conflicts, but was the psychoneurosis and the peptic ulcer patients.

Ulcer and the Psychosomatic Approach

Women subjected to emotional tensions and social hazards develop psychoneuroses and occasional gastric ulcer; men similarly exposed develop duodenal ulcers and occasional psychoneuroses. Such, of course, is a general statement subject to semantic criticism, but it brings up the subject of the differentiation and the dividing line between the psychoneuroses and the psychosomatic diseases. The emotional strains of life and its adverse contingencies will in the majority of its subjects give rise to fears, frustrations, functional disturbances, psychoneuroses and even some types of schizophrenias. The same conflicting emotional strains, carried out over a long period of time, will in a smaller number of persons, disturb the hypothalamic centers of control in the sympathetic nervous system and by disorganizing secretion, motility and tonus become so exaggerated as to result in actual organic disease. This is the modern concept of a psychosomatic disease, a psychic factor of disturbing force affecting a somatic or visceral balance so as to produce an organic change, in this instance

a peptic ulcer. It has been clearly shown by Keller (7), by Sheehan (8), and by others that the experimental interference with the nervous centers in the hypothalamus of monkey and dog will produce such disequilibrium of secretion, such spasm and congestion that the passage from functional disturbance to actual acute ulceration can be clearly demonstrated in the stomach and intestine of the experimental animals. Wolf and Wolff (9) in their superb human experiment on the exposed gastric mucosa of their human subject have demonstrated an identical end-result in the human mucosa. These facts line up with the earlier observations of Cushing (10) on the incidence of gastric lesions accompanying cerebral and cerebellar disease in man, as repeated by Mittleman and Wolff (11) and as again depicted more recently by Davidoff (12) in his series of clinically demonstrated brain lesions accompanied by gastric and duodenal ulcerations. Thus psychic emotional factors and/or organic brain diseases may, either one of them, produce the end-result of peptic ulceration.

That an organic personality constitution may underlie such a psychosomatic process is evidenced by the familial incidence of ulcer. Gastric and particularly duodenal ulcer may affect several members of one family in a single or successive generation; identical duodenal ulcers in identical twins have been noted (Boros) (13) and hemorrhage and bleeding ulcers in several siblings is a fact observed and of significance. The family history of ulcer patients, if obtainable, might have great bearing on insurability.

From the standpoint of insurability, gastric ulcers differ materially from duodenal ulcerations. Gastric ulcers are seen to occur with equal frequency in both male and female, though more recent statistics show a preponderance of males over females in the ratio of 2.1 to 1.0 (Bockus) (14). In gastric ulcers the average acidity titration is lower, materially so, than in duodenal ulcers, and night hypersecretion is usually absent. Gastric ulcers affecting females are subject to hormonal influences. Such ulcers usually heal during pregnancy, though they may recur shortly after parturition. They are likely to be exaggerated in their clini-

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cal course after the menopause. With gastric ulceration is associated the closely related problem of gastric cancer. The much discussed problem of how often does a gastric ulcer undergo malignant degeneration has, at least to my mind, been finally resolved. The mischief perpetrated by misinterpretation of the remarks of Wilson and MacCarty (15) of the Mayo Clinic (1910) has finally been corrected. They are said to have stated that 71 per cent of cases of gastric cancers showed sufficient histopathological evidence of previous ulcer to justify labeling them as cases of cancer developing on ulcer. This is an entirely different statement from one which implies that 71 per cent of benign gastric ulcers undergo malignant degeneration. Little by little this erroneous impression has been whittled down until today most clinicians would concede, at the most, an incidence of 2 to 5 per cent of such malignant degeneration. Actually I have seen but one case where a presumably benign lesser curvature ulcer followed over a course of years ended in a proven gastric carcinoma at the site of the ulcer (exploratory operation). Most clinicians and pathologists have resolved this thorny question of differentiation by conceding such a process of malignant change as an exceedingly rare occurrence.

But such a scientific concession or regression does not by any means resolve the question of how to differentiate clinically a gastric ulcer from a gastric malignancy when, in the middle or later decades of life a gastric ulceration is first observed. The question is not "Is this a change from a benign to a malignant status?" but is really "Are we dealing with a benign ulcer or with a neoplasm?" For in shortness of clinical duration, in subjective symptoms, in roentgenographic appearance, in secretory studies and even in gross and often in histopathological studies, the two appear almost identical. The clinical history and subjective complaints of the two diseases are not distinctive; secretory deviations from the normal are of no diagnostic significance for it is a well-established fact that normal acid curves are not inconsistent with carcinoma, the usually expected anacidity may not be present, and occult blood in the gastric content and in the

stool may be positive or negative in either or both conditions. Nor is the roentgenographic differentiation entirely trustworthy for a margin of error of approximately twenty per cent must be allowed. Certainly grossly when holding the specimen in the hand after resection, many mistakes are possible, and even the cut section under the microscope is liable to variable interpretations. Fortunately the gastroscope has become an instrument of greatest benefit in the scientific differentiation of benign from a malignant status and yet here too the ultimate word is subject to a margin of error. At times the X-ray errs and the gastroscopist corrects, at times the gastroscopist misinterprets or fails to see and the roentgenologist adjusts the error. Both methods are relatively reliable; neither is infallible.

The practical guide has been the clinical, roentgenographic and the gastroscopic course of the ulcer under medical treatment. If the ulcer heals in four weeks, disappears from the films, and the patient feels subjectively well, the ulcer was benign; if not, and it persists, it is malignant. But here too unfortunately even this last and ultimate criterion is subject to mistaken interpretation. For in the presence of a malignancy, the patient may gain weight and feel subjectively well, the roentgen films may show *almost* disappearance of the niche or defect, and gastroscopically healing of the ulcer base may be demonstrated and still the lesion is a neoplasm. Such an occurrence is not a rare clinical observation and is well explained upon the remarks and facts demonstrated quite recently by Palmer (16) in which he showed that a gastric malignant ulcer, under medical treatment may temporarily show granulation of the base of the ulcer, superficial epithelialization, and roentgen diminution just as does a benign ulcer under identical conditions of therapy. Again and again I have been deceived by the beneficial effects of medical therapy upon an ulcer, and have been lulled into a sense of false security for weeks or months only to observe recurrence of activity in the ulcer and the final establishment of a diagnosis of malignancy often too late now for lifesaving resection. Since the conditions of differentiation of benign from malignant ulcerations is so difficult it be-

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comes essential to urge resection of the gastric lesion where any possible doubt exists.

It takes years of clinical experience to postulate the difference between benign and malignant ulcer and then one is never sure of his own opinion. One approaches the subject at all times with fear and trepidation. Unless the lesion heals promptly and completely as demonstrated by clinical roentgenographic and gastroscopic means, unless the ulcer has been seen on previous occasions and is the type that runs the typical life-cycle of recurrence such as characterizes a benign ulcer—otherwise, every gastric ulcer of short duration in the middle-aged should be resected. The insurability of a known gastric ulcer is probably a question that should be answered in the negative.

On the other hand, gastric ulcers lend themselves well to operation, the mortality of such a procedure is low, the rate of recurrence as a marginal or stomal ulcer is practically negligible. Post-operative anacidity, particularly in the female, is almost the rule and later recurrence is exceedingly exceptional.

The *DUODENAL ULCER* presents a different set of problems. Here we are dealing with men, with high post-prandial acidity titers, with continuous night secretion of low Hion concentration, with an obstinate rate of recurrence under medical treatment. The psychic and psychosomatic aspects of duodenal ulcer are very evident, the emotional and physical travail of life complicate the long-range problem. Malignant degeneration of duodenal ulcer is of course out of consideration. On the other hand, the operative procedure is complicated by the frequent penetration of the ulcer into the head of the pancreas, the difficulty of closing-over the stump of the resected duodenum, the tendency to post-operative leakage. While the overall operative mortality is low (4.26% to 1.5%) the rate of recurrence is highest in duodenal ulcer resections due to the tendency of at least one-third of the cases to persist in maintaining, after the resection, a materially high post-operative gastric secretory acidity.

Complications of Peptic Ulcer

Little change has occurred in modern viewpoints as regards the complications of ulcer. Perforation takes place in perhaps 1 to 2 per cent of ulcer cases. Pyloric stenosis is somewhat more frequent. It is of interest that the conservative treatment of partial stenosis is frequently favored. Not every stenosis is an immediate call to surgery, many of the incomplete obstructions yielding to daily emptying (no lavage). If after several repeated nightly evacuations of stomach residue the symptoms subside and vomiting ceases; if the residues removed are successively smaller each night, then the case is considered favorable for medical treatment. If the residue remains material in quantity then operation is indicated. After a study of the plasma electrolytes, after restoring the normal values for chlorides and proteins, after eliminating the alkalosis and the restoration to a normal CO₂ combining power coefficient, then a subtotal gastrectomy rather than gastroenterostomy can be performed with safety. To make assurance doubly sure a jejunostomy for post-operative feeding is of considerable value.

As regards hemorrhage, there are in recent years two viewpoints or factors which rate discussion. One is the increasing assurance with which hemorrhage from peptic ulcer can be treated successfully. The former high mortality figures for even massive bleeding are no longer encountered in the literature. The figure of 6.5 per cent mortality which we (17) published in 1939 represents about the average for most publications of recent years. This figure was bettered by Marshall and Kiefer (18) (4.6 per cent) while on the other hand Blackford and Cole (19) indicated a mortality figure of 16.8%. These latter percentages are, however, hardly comparable since they cover only cases of extreme massive hemorrhage and arbitrarily exclude all the more moderate degrees of bleeding. Kirsner and Palmer (20) noted a mortality from hemorrhage of 10.6 per cent between 1929 and 1939, reduced in their more recent series to 3.47 per cent.

The most interesting recent development is the introduction of liberal feeding during the actual hemorrhage by Meulengracht in

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1937. The older dietary treatment during the hemorrhage called for complete abstinence from all foods, a view to which I still personally cling. However, the younger generation of clinicians follows mostly the dictum of Meulengracht who allows free and liberal feedings, even of meats, during the actual phase of bleeding. With such liberal dietary freedom Meulengracht claims a lowering of the mortality of hemorrhage, to 1.8 per cent, a very promising figure indeed. However, Schiff (21) who favors liberal feeding could not reduce the figure for mortality below 6.8 per cent, which is exactly the figure which we published 15 years ago for fasting during hemorrhage. All in all, hemorrhage from ulcer seems of increasing incidence (10-25 per cent), particularly since and during the recent war, and yet conservative treatment and transfusions have to a large part removed the fear of this phenomenon as a complication of ulcer.

Medical Conservative Treatment of Peptic Ulcer:

The overwhelming percentage of ulcer cases are still reported as medical, and not as surgical problems, only perhaps five per cent requiring operative intervention. Even though we now realize that ulcers under the very best medical regimen have a strong tendency to recur and to recur throughout a lifetime, even though we speak of "cure" of ulcer with mental reservations because of its tendency to chronicity and recurrence, we still prefer medical treatment. Modern methods of conservative treatment show great advances over the older Lenhartz or Sippy forms of management. The diet is likely to be more liberal; the addition or even the replacement of diet by amino acid preparations (casein hydrolysates) probably represents a distinct advance in dietary therapy. While the period of observation is entirely too short to allow of a final estimate, the neutralizing power of amino acid concentrates, their protein content and easy assimilability gives promise of rational utilization.

The colloidal aluminum hydroxide products as a substitute for the alkalies, marks an advance in therapy. The intragastric milk drip and the continuous intragastric drip of cremalin and amphotogel serve well and successfully to terminate pain promptly

and bring about rapid amelioration of symptoms and cure of the ulcer base. This last and newer method of treatment has not received the popular medical acceptance that it truly warrants. The relief of ulcer symptoms, particularly pain, is very prompt and gratifying. Though the end results, in a long range follow-up may not warrant its regard as a permanent cure, and though recurrences of the ulcer may later occur with disappointing frequency, as a method of immediate surcease from pain and distress it is without an equal.

Surgical Treatment of Peptic Ulcer:

Five per cent of my own cases, fifteen per cent of the ulcer patients at the Mayo Clinic, are considered to be candidates for surgical intervention. The operation of choice is today subtotal resection, that is removal of a half, preferably two-thirds of the lower segments of the stomach with a jejunal anastomosis to the stump of the stomach (Hofmeister, Billroth, Balfour type of anastomosis). The duodenal ulcer itself should by general acceptance be removed, since otherwise a gastroenteric stomal ulcer may recur at or just distal to the anastomosis. At this point opinions vary; many surgeons of experience, such as Lahey (22) and Lewisohn (23) and Hinton (24) insist upon the removal of the ulcer base even though the ulcer penetrate the head of the pancreas. By so doing the operative mortality may be somewhat increased but the likelihood of recurrence is diminished. Others, such as Colp (25) and Heuer (26) occasionally avoid the inclusion of the ulcer base in their resected specimens when by so doing they are likely to invite post-operative leakage and infection and so materially increase the operative risk. In the poorer risks, gastroenterostomy, in preference to resection is still practiced even in a material percentage of the cases, though the likelihood of a recurrent gastro-jejunal ulcer is thereby increased (roughly 15-25 per cent). The operation of gastroenterostomy should be restricted to advanced cases of pyloric stenosis or to such extreme surgical risks that the lesser, rather than the greater operation seems a valid choice. At that, it is a question

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whether gastroenterostomy is ever preferable, since in the recent statistics from the Mayo Clinic the operative mortality from gastroenterostomy (1.5 per cent) was practically identical with that of subtotal resection (1.6 per cent) (27).

It is a remarkable fact, one worthy of great attention that the surgical mortality from gastric resection for gastric or for duodenal ulcer has been successively and successfully reduced from its original risk of fifteen per cent, to nine per cent, five per cent and in recent years to 1.6 to 4 per cent. The recent figure published by Lahey (28) of 1.5 per cent is truly astonishing. This beneficial elimination of all but the most grave risks in operation can be explained on several grounds. Much better and lighter anesthetics such as cyclopropane, ether and oxygen, intravenous pentothal, (supplemented by intravenous curare) as an adjuvant, continuous intra-spinal anesthetics of pantocaine—these have been great factors of safety. The better preparation of the patient before operation, lavage, intravenous saline and glucose, transfusion during the actual operation and thereafter to anticipate shock and to overcome it, have acted as increasing factors of safety. The post-operative complications of pneumonia and abdominal infection are today met by anticipatory courses of penicillin as a routine post-operative method. The Levine tube, the Wangenstein drainage overcome post-operative atony, one of the most distressing of late complications. In this manner surgical mortality has been reduced almost to the vanishing point. In fact, in my own practice and experience in the last eight to ten years, I have been completely free of operative disasters so that one approaches gastric resection for ulcer today not with the fear and trepidation of former years but with an honest assurance to the patient that he may truly expect to survive the operation and to be and to remain cured.

A new and interesting development is entering into this field of gastric surgery for peptic ulcer. In 1943 (29) Dragstedt of Chicago recommended a new type of operation for peptic ulcer,

a procedure that consisted of the transthoracic resection of both vagus nerves in the chest, a supra-diaphragmatic approach. The infra-diaphragmatic resection of the anterior vagus nerve at the cardia had been practised as long ago as 1923 (30) by Winkelstein and Berg but the effects were equivocal and the suggested procedure never gained credence. Dragstedt and his colleagues by resecting both vagi above the diaphragm sever the main stimulatory nerves that control the cephalic phase of gastric secretion and by so doing eliminate the psychic factor, the psychosomatic influence that controls a large part of gastric acid secretion.

They had operated upon 38 cases by 1945 (31), many more since, and claim cure of the ulcer and of its symptoms in the very largest percentage of their cases. The drawback is an unusually severe and protracted gastric atony which frequently if not usually results from vagus resection, an atony which at times becomes so severe as to require a secondary gastroenterostomy. Where a previous gastric resection or a gastroenterostomy had been performed, and where vagus resection is practised because of recurrent anastomotic ulcer, the atony is automatically provided for and the operative result is usually entirely satisfactory.

The claims of Dragstedt have been substantiated by Moore and Jones (32) and their group reporting from the Massachusetts General Hospital, by Ruffin (33) from North Carolina, and by Colp (34) and his group at Mount Sinai in this city. As a cure for and complete relief of gastro-jejunal ulcer this procedure has no equal, as I have witnessed in several of my personal cases. The most interesting fact is, that in spite of so major a procedure as a trans-thoracic incision covering an area of eight ribs, and a trans-pleural approach, the mortality of the operation is practically nil and the subjective improvement is immediate and complete. Whether this new surgical procedure will completely replace subtotal resection for primary ulcer remains yet to be established. Certainly it is no substitute for anastomotic procedures in cases of pyloric obstruction; but for high lesser curvature

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gastric ulcers and for duodenal ulcers with patulous pylorus it may offer a safe and permanent cure of lasting durability. As a cure for recurrent anastomic gastro-jejunal ulcers the bane of previous surgical procedure, obstinate, life-threatening and incurable by any number of subsequent gastric resections, supra-diaphragmatic bilateral vagus resection has added a great step in the advance of gastric surgery for benign peptic ulcer.

The Insurability of the Ulcer Patient

Given a co-operative patient, one who can and will follow a dietary restriction, one who will abhor cigarettes and avoid strong alcoholic drinks, one who will guard against excessive mental and emotional problems and conflicts, then we can carry an ulcer patient for a lifetime with a promise of little or no loss of working efficiency, small likelihood of complications and a minimum likelihood of losing his life from any immediate cause related to the ulcer. Let us look at the overall picture, the ultimate life prognosis for the ulcer man or woman. From the lesion itself, disregarding for the moment its complications, the ulcer patient need never succumb. Hemorrhage occurs in perhaps 10 per cent of the cases and has a mortality of 6 to 8 per cent; that is, 0.6 to 0.8 per cent of all ulcer cases may die of hemorrhage. Perforation is rare and occurs in perhaps two per cent of cases; the operative mortality is 10 to 25 per cent. That is 0.2 to 0.5 per cent of ulcer cases may die as a result of perforation. The ratio of the sexes in perforation is approximately 25 to 29 males to one female. In the large series published by Sallick (35) (74 cases) no females were encountered. Both in hemorrhage and in perforation the female ulcer case is relatively secure from a fatality.

Pyloric stenosis is easily countered by medical means or at worst is cured by surgical intervention with a minimal risk.

Five per cent of peptic ulcer cases are operated upon with a mortality figure today of 1.6 to 4.0 per cent. Taking the larger figure, the total mortality of all ulcer cases as a result of surgical

intervention will be 0.2 per cent. By combining all these figures we note:

MORTALITY ESTIMATE

Uncomplicated ulcer	± 0
Hemorrhage	0.8%
Perforation	0.5%
Surgical mortality	0.20%
Total expected mortality	<hr/> 1.5%

In other words, the life expectancy of the ulcer case is impaired, in comparison to the normal life expectancy of the control population, by about one and one-half per cent.

In contrast to diabetes, to rheumatic heart disease and similar chronic maladies, the ulcer case offers a low rate of risk. The high incidence of ulcer in the general population, 5 to 10 per cent, creates for the actuary and for the medical director of life insurance companies, as it did for the military branches of the government, a material problem. But the overall small risk to life offered by the ulcer applicant for insurance compensates for the magnitude of this number and brings assurance that the risk to life involved by the disease is in inverse proportion to the widespread distribution of the malady.

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PRESIDENT STREIGHT—Gentlemen, we have another excellent chapter added to the transactions of our Association. Thank you very much, Dr. Crohn, for your very interesting paper. This paper is now open for discussion.

DR. J. M. LIVINGSTON—I would like to ask Dr. Crohn a question. These tests, which are over a long period of time, don't correspond with his explanation. We only get the bad risks that have duodenal ulcers. The only people who have duodenal ulcers are the bad ones. Are the good ones left outside?

DR. FRED W. ROLPH—I, too, would like to ask the speaker, do ulcer cases that have a history of hemorrhage have a greater mortality than those that have not?

PRESIDENT STREIGHT—Are there any other questions now? Would you care to reply to that now, Dr. Crohn?

DR. CROHN—I wish the insurance companies would supply us with more statistics on the subject of ulcers. I don't know of any, outside of the Metropolitan Life Insurance Company's statistics. I am very anxious to have the experience of the insurance companies on ulcers. I am sure there are, buried in the archives of statistics of the insurance companies, a great many answers to the problems we would like to know. I don't know whether insurance companies get a fair sampling of ulcer cases as we see them in clinical life.

As regards hemorrhage, the point Dr. Rolph spoke about, it is true that the man who has had one hemorrhage will likely have subsequent hemorrhages as he gets older. After he passes forty-five each hemorrhage becomes more dangerous to life than it would have been previously. That is why we allow a patient one hemorrhage and send him off on parole. After the second hemorrhage he becomes a candidate for surgical treatment, but the subsequent hemorrhages are usually less severe than the first one. Subsequently, the hemorrhages are liable to be less severe. As I say, when a man persists in hemorrhages, shows more than two hemorrhages, he is a surgical risk and should be operated upon.

DR. JOSEPH W. JOHNSON—I was very glad to hear Dr. Crohn's histologic ideas agree with mine. I think this subject of vagotomy is going to play an important part in insurance risks. They have done about twenty cases in Toronto

with very good results. Take, for instance, the case of a young man who won't pay any attention to diet. After he leaves the hospital he gets a return. I think that is the type that it is going to be very valuable to help. I was also interested in what Dr. Crohn said about the supposition that used to prevail, that there was a greater mortality among the white collar class. For some years now, I have made it my business to see all the returned medical missionaries, to see what recurrence of ulcer there was in widely separated places, such as China and Abyssinia, and they say that it is just as frequent in those countries as it is here. It is not diagnosed or treated as such, but as far as they can see in their hospital work in these countries it is just as frequent there as it is in the United States. I would like to thank Dr. Crohn for his paper.

DR. WENSTRAND—It so happens that I listened to Dr. Dragstedt a week ago last night at the Milwaukee Academy of Medicine and it was a most interesting experience. His cases now have risen to 125, and he made the definite statement that they were all cured. He had only one dead and that patient died from pneumonia, and he also had overcome the lesion in the stomach by using the fine tube in the stomach for three or four days after the operation.

DR. CROHN—As regards the incidence of ulcers all over the world as reported by returning missionaries, that is very true. We were told that the Japanese lived on rice and that they, therefore, had no nerves; we have been told that the population of the South Seas are an easy-going people, they like to play the guitar and sit in the sunshine all day, so they had no ulcers. But when you got down to statistics and compared them it was proven that in all countries they have ulcers, and that ulcers are practically the same everywhere. Here it is a disease largely of the white collar and professional people. Andy Rivers said twenty years ago that 20% of the doctors had ulcers. Andy Rivers was sitting in a medical meeting in New Haven when he said that many years ago, and somebody

said, "I would like to confirm that right here in this audience. Would it be possible to take a poll and find out how many doctors present have ulcers?" And so they polled the audience and it was exactly 20%. I don't want to try it here. (Laughter.) I won't embarrass you. Does the surgeon solve the problem of ulcer by cutting off the somatic nerve? No, I think that concept is something you must understand about psychosomatic diseases. I think it is the psychology that originates the disease, but once it is formed it is an organic process and goes on, whether you reverse or continue the autonomic strain.

Take the ulcer case—there is no more psychological patient than one who has been subject to severe somatic strain or continued strain, and ulcer starts, and the patient shoots a temperature of 103 and develops diarrhea. A great many people think all you have to do is to send for a psychoanalyst and reassure the patient to be quiet. The ulcer is there in the intestines. It is impossible by sending for a psychoanalyst to help that patient, but I have seen that done again and again. You can't cure that patient in that way. It is psychology that starts the disease but once the disease is started it is going to run its course. The patient may get perfectly well. I have seen that repeatedly. For instance, a woman stands on the curb and sees her child under an automobile. The child isn't touched by the automobile, but within a few days this woman will have a complete recurrence of ulcer. It is there that the psychologic condition comes in again, but you cannot reverse the disease by sending for a psychoanalyst, nor think you can cure an ulcer by cutting the autonomic nerve, once the ulcer is there. What the cutting of the vagus nerve does is to stop secretions that produce acidity.

PRESIDENT STREIGHT—Our next subject is "Veterans as a Medical Underwriting Problem" by Dr. J. Raymond B. Hutchinson, Medical Director of the Acacia Mutual Life Insurance Company. Dr. Hutchinson!

VETERANS AS A MEDICAL UNDERWRITING PROBLEM

BY J. RAYMOND B. HUTCHINSON, M. D., MEDICAL DIRECTOR
ACACIA MUTUAL LIFE INSURANCE COMPANY

In the underwriting selection of the veteran there are many important diseases to be considered. Much has been written about most of these and many papers have been read within the last year. Therefore, I will not go into any great detail but will touch on the common conditions which may become problems to us in the medical selection of the veteran.

I was fortunate that my duty assignment in the Navy was exclusively in the Bureau of Medicine and Surgery where I was the officer in charge of and responsible for the processing of medical surveys. These were the personal medical reports and recommended disposition of each hospital case by the medical staff of that particular Naval hospital. Over a period of some three years my organization handled almost 400,000 of these surveys and consequently my thoughts relative to the underwriting of our discharged service personnel, to a great extent, is based upon my observation while in this position.

I feel that one of the problems confronting the Medical Director today is the proper evaluation of a class of veterans' cases which we term repatriates and former prisoners of war. There were thousands of these cases both in the European and Far Eastern theatres. These people are now applying for life insurance and while it is probably a fact that no one Medical Director will handle any great number of them, there will be sufficient cases, if not carefully underwritten, that can very definitely form a block of underwriting which could be considered selection against the company. Consequently, what are we to do with such applicants? Let's look at the reported general facts which are common to this group as a whole. It is found that most of them when first seen by medical per-

sonnel were manifesting the most distressing symptoms of malnutrition. A constant finding was the marked abdominal distention such as seen in the enormous rice belly. Marked edema of the extremities was also present in between 30% and 50% of the cases reported. It should be stated that the edema was particularly prevalent during the change over from a starvation diet to a normal diet, which is not too well explained.

It might well be that myocardial, hepatic, or nephritic damage or any combination of these factors could have been the underlying basis for the amount of edema observed. On the other hand, it could have been also that the edema was, as stated above, likely dietary in origin.

There were neurological changes including gross damage to the central and peripheral nervous system. Complaints of numbness and tingling such as paresthesia, particularly of the extremities and back, impairment of vision and some disorientation were noted.

Considering the hardship experienced by this group the general mentality is not too abnormal. However, these persons represent the survival of the fittest. The rest are dead, not having either the mental capacity or physical stamina to survive. It is reasonable to assume that the duration and severity of their experiences will leave residuals of varying degrees.

The gastro-intestinal tract of these repatriates and prisoners probably suffered more than other parts of the body. Better than 50% of these patients that were under observation suffered from diarrhea. Amoebic and bacillary dysenteries were particularly prevalent; however, strange as it may seem, very few of the military personnel appear to have been affected by it to the same degree as the civilian. If figures were available, I am confident that they would show this to be a fact. A predominant finding in most all of these people was the constant presence of marked anemia. I am advised by some of the men supervising these patients that such cases of diar-

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rhea were not unlike sprue, and while under normal conditions the vast majority recovered, we must not lose sight of the fact that many have been incapacitated as the result of this terrific diarrhea for months and months. Although I would not attempt to go into the pathological aspects, the severity and duration of a condition of this character must have materially affected the liver and bone marrow. Whether this damage is irreparable or not, only study and observation can determine, particularly the medical significance from an underwriting standpoint.

In the above I have briefly summarized some of the most common diseases the bodies of our war prisoners and repatriates have undergone. With this in mind, the underwriting of applicants represented by this class becomes quite difficult. Personal experience in my company indicates we have had a considerable number of these cases for underwriting consideration. At the present time they present, in general, satisfactory physical appearance, but the medical history submitted indicates the existence of the above symptoms as recently as 6 to 18 months ago, many admitting that they suffered from enlargement of the liver during this period. Consideration should be given to the future significance of such findings as far as medical underwriting is concerned. We, of course, are aware that the physical condition of many, after due care, was considered satisfactory enough for return to full duty in the Service. Subsequent to this return to duty, many of them were retired. This service personnel represents the most satisfactory type, for in most instances they received better treatment while imprisoned and subsequent to their release have in general received better medical care than the civilian population. Considering the over-all picture of the prisoners of war and repatriates, it would seem reasonable to assume that organic damage has resulted from the previously described findings and to what extent it is reparable can only be answered by time and observation. My approach, frankly to the underwriting of these cases is one of caution, and probably

a waiting period of 3 to 5 years would be desirable in order to determine if there are any fixed residuals.

Acute Infectious Hepatitis

This problem is appearing more and more in literature today. We know that the condition, according to history, is associated with wars. It was very prevalent during the Civil War, running into thousands of cases, and in World War I running into tens of thousands, while in World War II the number would probably run into several hundred thousand. It is thought to be the result of filth and it can be considered as a pandemic infectious disease. In considering the disease we think of it in terms of infectious hepatitis with the etiological agent in all probability variable strains of a filterable virus. The so-called serum hepatitis following the administration of plasma is probably a sub-group of this same category. The marked contrast between the relatively few cases in the Navy, where good personal hygiene and food was possible, compared to the high incidence in the Army, where the men were exposed to filth, poor sanitation and variable food, in many instances while in combat, is interesting.

The recurrent nature of this disease in a yet unknown percentage has only recently become known. The remote sequelae resulting from progressive liver destruction is only now becoming apparent. I feel, however, that there have been a sufficient number of these cases to warrant giving this material study as far as underwriting selection goes.

We find that command after command in the various theaters of war were disabled up to 50% of their strength. In considering these cases as an underwriting problem we must realize that the majority affected were in the younger ages, namely below 35. I viewed these cases with liberality upon my first return from the Service; however, after giving considerable time to studying the entire situation, needless to say, I changed my opinion for I was more or less of the thought that this was a simple catarrhal jaundice and had not con-

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sidered the fact that it should be looked upon as an insidious recurring disease which could produce marked liver damage.

The infectious type of hepatitis was self-limited in about 80% of the cases, with disability for the service personnel reported as existing from 8 to 10 weeks. While the major portion of these people apparently recovered satisfactorily and were placed on a return-to-duty status at some military areas, I understand that in other army areas no attempt was made to return these men to active duty in light of the frequent relapses. It is reasonable to suppose that such relapses produced additional liver damage and that chronic liver disease could develop as a result of prolonged and recurrent attacks. Observations indicate that certain of these cases develop toxic necrosis of the liver cells and eventually cirrhosis of the liver.

Inasmuch as hepatitis may develop in a somewhat quiescent stage in which present symptoms are insufficient to interfere with the person's physical activity, we may find when they are presented as applicants for life insurance they are carrying on the duties of any average normal individual. A finding of slight periodic jaundice should be looked upon as one of the flags of a latent condition.

In the process of considering the acute infectious hepatitis as an underwriting problem of veterans, we must not overlook the serum type which I have been advised is more prevalent than we first had thought. While this paper refers to the underwriting of veterans, the so-called serum type of hepatitis may not only be a problem with veterans but with our non-veteran civilians. It is stated that one of the probable sources that may be responsible for spread of this condition is the collecting of blood plasma in large quantities. It has also been reported, following careful scrutiny of various methods of intravenous technique, that improper sterilization and autoclaving of instruments and needles used in this connection may be considered as a source of infection. I am told that this thought developed as the result of careful study in the British Medical Corps during this war.

This form of hepatitis is of a far more latent type, having an incubation period of as much as 60 days. In considering these cases as a whole, the thought might have occurred that most of the symptoms produced are the result of varying degrees of liver deficiency, which, in turn, are the result of a certain amount of destruction of the liver proper. Since there exist varied opinions as to the number of these cases that recover completely, it would appear proper, when evaluating these risks to consider quite carefully the preceding history. While we know that the liver regenerates itself, the fact remains that there has been liver damage as the result of inroads of infection. This raises the question as to just how far to go in selection. Autopsy has revealed that there is an enlargement of the liver in 60% of the fatal cases, which is undoubtedly due to some intrahepatic pathological process. Studies have been presented supporting the view that the lesions in the liver of the fatal cases correspond to what is long known as idiopathic acute yellow atrophy. In fact, one pre-eminent authority states that in all cases which came to autopsy, without exception, the liver changes were typical of idiopathic yellow atrophy.

The Medical Director at present is faced with making an equitable decision as to the selection of cases of this character. With such a known picture, it would appear that we should approach the underwriting of these cases with considerable caution until more experience has become available.

Rheumatic Fever

In passing, a word should be mentioned about the selection in connection with rheumatic fever. While we have very specific ratings for this disease, the thought could be given that the cases of rheumatic fever incurred in the Service were not only picked up in their very early stages in most instances, but were under such careful medical scrutiny that those who have recovered satisfactorily could probably be considered with more lenient ratings than the usual civilian case which we

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underwrite. While I have no figures to support this opinion, I did have the opportunity of observing the methods, from its inception, of the manner in which the Navy handled their patients down to and including their rehabilitation procedure. There were a large number of these cases that had been diagnosed early and handled properly from the onset of illness, recovered satisfactorily and were returned to full and active duty. There were several hundred of such cases which I was following, but due to demobilization any possibility of ever obtaining the end results was prevented. Due to lack of specific information concerning the beneficial effects of the newer types of therapy used in the treatment of rheumatic fever, we must, therefore, depend upon our present methods of selection until concrete evidence is available.

Tropical Diseases

Mention might also be made of some of the tropical diseases such as malaria, the dysenteries and filariasis. I do not believe that there will be any post-war problem as far as the underwriting of these diseases is concerned. Much has been written since I gave my first opinion on the matter which was in 1944; however, I still am of the mind that we can be guided by our usual ratings for the dysenteries and malaria in practically all instances. Those that present more trying clinical problems would not be insurable anyway. I have always taken the position that filariasis was insurable and insurable at standard rates. The very careful studies made by Dr. Coggeshall during the time he was responsible for the rehabilitation of these cases for the Navy and Marine Corps certainly support the opinion that they can be underwritten with security. Within the last 3 weeks I have heard the statement made by one of the leading students of epidemiology in the Navy that it is thought the disease will burn itself out within 2 to 3 years completely, while 1 year to 18 months ago it was thought it would probably take from 8 to 10 years.

Psychoneurosis

No paper would be complete in considering the underwriting of veterans without some comment concerning psychoneurosis. In 1944 I made some very general statements as to the acceptability of psychoneurotics for life insurance. Subsequent to the close of the war, I made further study of this matter and with approximately one year of practical application in our underwriting department, I have come to the conclusion that there should probably be a more liberal interpretation given these cases than we have heretofore.

However, to digress for a moment, let us consider why and when certain people may have become psychoneurotic during the recent national emergency.

First, as to *why* this condition became evident:

- (1) Large numbers of those procured for military service were in that age of youthful immaturity where their decisions and actions were influenced to a considerable degree by their emotions.
- (2) Our environment and psychology of the past twenty years has not been conducive to the prosecution of war.
- (3) The abrupt onset of war necessitated marked environmental readjustment. This situation, coupled with the stress and strain of military regimentation and its training programs, placed severe demands upon the nervous system of those involved and resulted in the inability of large numbers to make a satisfactory adjustment.
- (4) Another group performed militarily quite satisfactorily until exposed to combat or pre-combat conditions, at which time the effect of one or more of these above mentioned factors became evident.

Secondly, as to *when* this condition became evident:

- (1) Failure to meet the required military standards for entrance into the armed services. These cases have arisen or will arise following the rejection for induction or voluntary enlistment. The underwriting problem

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here resolves itself into distinguishing between the immature youth whose emotional balance was reflected in his conduct, that is, circulatory, neurological, gastrointestinal manifestations, etc. and those who were mal-adjusted to society in general. The former could probably be and are underwritten upon a more favorable basis.

- (2) Failure to adapt to the regimentation and stresses of military life during the training period. It is suprising when we consider the number that were found not temperamentally adapted and released from the Service during the training period. Most of those returned to civilian life for this reason have made a satisfactory readjustment, and I feel that they will present us with little difficulty from the standpoint of selection as insurance risks.
- (3) Failure to endure the hardships of actual combat. Various terms such as combat fatigue, battle fatigue, etc. have been used in referring to this group. This condition did not appear in most of these persons until the individual was placed in an abnormal environment. The threat of survival had alerted his protective reflexes and basic instincts, and frequently physical hardships had weakened his moral fiber.

The early recognition, prompt and effective treatment, with rehabilitation measures based on a proper understanding of the basic causes has resulted in recovery in the majority of these casualties. One of the notable contributions of the medical profession in this war is the record of such cases that have recovered and had been returned to civilian life in a useful capacity.

I would like to add, however, just one word of caution concerning certain of these individuals who are experiencing various adjustment difficulties following return to the role of a civilian. Observations to date indicate that we should be a bit cautious concerning the veteran who, following six months

resumption of civilian existence, is still showing readjustment difficulties. True, in some cases, circumstances of hardship and insecurity, the results of present domestic conditions, warrant individual consideration; nevertheless, I believe the greater the time interval required to readjust in excess of six months, the greater should be the caution when this individual is considered as an insurance applicant.

Having briefly outlined my concept of the psychoneurotic in so far as what readjustment problems he may present from an insurance standpoint, let us consider the future of those who are mentally unimpaired but suffered permanent physical deformity and from such deformity will be handicapped to a greater or lesser degree. These persons will benefit by readjustment training, even while still in the convalescent period, for rehabilitation programs, as you know, include occupational therapy, training in the use of markedly improved types of artificial limbs, and schools of education for the deaf and blind. Later, further benefits of college education or on-the-job training are available to these people. This training, plus the benefit of disability pensions and the services of the expanding veterans' facilities, should materially aid in the readjustment of these unfortunates. Time alone can determine to what degree and in what measure this program will be successful. Our experience following World War I may prove helpful, but the picture today contains many dissimilar factors which must be considered.

And now, what of the 8 or 10 million veterans discharged who do not come under the heading of psychoneurosis? Every veteran in general who returns to civilian life, we might say, is still mentally and nervously organized for war. The passage of time and proper associations are needed for the correction of this condition. The veteran must give up his attitude toward civilian life and form a new one relative to his changed situation, and the sooner that he does this, of course, the quicker will be his adjustment. The veteran returning from the service many times cannot except the discipline of peace. He is

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used to being dependent upon others for guidance, instructions, and maintenance. He is hampered in finding a position because many of these men, before their service careers, had no civilian position except being youngsters out of high schools and colleges. The veteran has dreamed of travel, of success, and the most ambitious have the most grandiose plans as to their own personal ability and to the future. Civilian life looks easy to them when it is in reality actually very hard for the veteran. There is the waiting and the starting of things at the bottom, the beginning all over again. There is the restless urge to give things a push to hurry them up, to play for high stakes, to do something spectacular and thus make up for the time this man or woman has lost in the service of our country while other civilians have apparently outstripped them. When these things are not accomplished, there is bitterness and disappointment, and such is evident in their conduct and conversation. We do not consider this as abnormal, nor do we penalize them as such when applying for life insurance. In what manner these factors will enter into effecting a satisfactory solution of this present problem of many, will depend largely upon what is being done and what will be done to solve the problem of insecurity.

These people went out and fought to establish security, returning home they find insecurity such as domestic unrest, and shortages of the essentials to which they were accustomed before going to war. How successful the solution of these problems will be with the resultant satisfactory readjustment will depend to a great extent on the manner in which these issues are solved. If such are not solved satisfactorily, we have in this vast reservoir of people the elements for producing an additional number of psychoneurotics.

Considering all of these factors, the intelligent selection and underwriting of this group will rest with the Medical Director. It concerns itself as an individual case problem for the time being and each case should be underwritten on its own merits.

Incidence of Coronary Disease at the Younger Ages

Another phase of the underwriting of veterans which undoubtedly has recently come to the attention of all of the Medical Directors is the incidence of coronary disease at the younger ages. We read in our periodicals recognition of this fact. It brings forth some interesting problems of underwriting which do not necessarily confine themselves exclusively to our veterans. Formerly, as far as diagnosis was concerned, we believed coronary artery disease was most prevalent in the upper ages, and when I say upper I refer to the sixth and seventh decades and hypertension was considered to be one of the most frequent causes of the disease. We now find that it is reported as occurring more often in the younger ages than formerly, noteworthy in the fourth and fifth decades and even down to the third decade. The condition is most frequently associated with stress and strain.

During my period of service with the Navy, we dealt principally with the men of the younger ages and early in the war we heard of men in combat suddenly dropping dead of attacks that simulated coronary artery disease. This was first brought to my attention by a battalion surgeon of one of our Marine Corps Amphibious Units. His statement was that there had been deaths in his battalion which he felt sure had resulted from coronary occlusion and these men were in their late 20's and early 30's. There were no post-mortems to prove this and his conclusions were based entirely on symptomatology.

As I recollect, there were a large number of deaths from coronary thrombosis and most of these deaths probably occurred in the fourth decade, in view of the fact that the average age of men in the Naval service was around 35 years. It was noted there were many men discharged from the service with coronary artery disease who had no symptoms or evidence of the condition until placed in the position of stress or strain which, of course, included mainly combat. As a cardiac consultant for the Veterans' Bureau, I have had some cases within the past year referred to me whose histories were typi-

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cal of coronary artery disease. Two cases in particular stand out in my mind, both men under age 30, in active areas of combat. One was on duty with an engineering unit just behind the front line and was suddenly taken with acute substernal pain. He became breathless and went into shock. The battalion surgeon diagnosed this condition as coronary occlusion. Facilities for electrocardiographic diagnosis were not available. Subsequent to this period, cardiographic tracings taken indicated coronary occlusion of Class 2 type. Currently, the man has no symptoms. His examination findings were negative other than the electrocardiographic tracing which showed there was some coronary artery disease with myocardium damage. The 4th lead did not give any indication of an old myocardial infarction.

The second case was that of a man in active combat who noted anginoid pain but did not turn in to his battalion surgeon until several hours later. His symptoms were substernal pain, with pain radiating down both arms and a tingling sensation more pronounced in the left hand than in the right. He was hospitalized and later invalided from the service. He advised me that he had had rheumatism as a boy but no one had ever informed him that his heart was impaired. He had passed all of his draft board and army physicals and it was only after turning in to the battalion surgeon that a diagnosis of heart disease was made. Both of these men at the present time are actively engaged in laborious work. They looked healthy and quite acceptable for life insurance were not these histories revealed. I mention these cases not with the thought of presenting anything clinical but to emphasize the effect of emotional disturbance on the heart. We know that such disturbance is not uncommon for it has been recognized innumerable times during emotional strain. The Medical Director in an attempt to speculate as to the effect of the emotional stress and strain upon our millions of veterans who have been adjacent to or in contact with combat should give due con-

sideration to the resultant effect of such on the circulatory system.

In closing, I feel it should be pointed out that the various underwriting problems suggested in this paper are common every day problems that pass over the desk of the Medical Director. It is not my thought or proposal that the veteran should be discriminated against and considered an actual underwriting problem any more than any non-veteran civilian who is applying for life insurance today. However, we have gone through some abnormal times where there have been abnormal conditions and out of it all have developed, shall we say, a greater number of possible substandard or declinable risks than we have had heretofore. I, therefore, have pointed out as I saw them some of the underwriting FLAGS which I hope will be useful in our future handling of these cases.

President Streight—The discussion will be opened by Dr. Archibald C. Wilson of the Connecticut General. Dr. Wilson!

Dr. Wilson—During my tour of duty with the Navy, I had the good fortune to be associated with Dr. Hutchinson. It is therefore a particular pleasure for me to be afforded this opportunity to discuss his paper.

Dr. Hutchinson has reviewed several service-connected diseases, and has pointed the way to the underwriting approach we should take on applications presented to us with such histories. Important as they are, time does not permit me to comment further on all of the conditions which he has discussed.

It is estimated that 30% of all medical discharges during the war years were due to psychiatric conditions, to say nothing of the vast numbers who were rejected at the preinduction stage and by aptitude boards during the first few weeks of training. Dr. Hutchinson's paper referred at some length to this large class. It seemed to me that the most constructive way I could comment on his paper would be to enlarge upon his remarks and go into the interpretation of the psychiatric discharge, for underwriting purposes.

The first point I would like to make is that upwards of 50% of all of those psychiatrically discharged were only mildly emotionally unstable. In civilian life they might have been regarded as "nervous," as "worriers," or "somewhat neurotic" by their friends and family physicians, but few indeed would ever have seen a psychiatrist or acquired a psychiatric diagnosis in civilian life. You would have accepted them for standard insurance without question. Why, then, were they discharged? Without going into all of the reasons, the fact remains that they did not, and could not, meet the demands of a modern military organization. You must remember that they were living in what, for most of us, is a highly abnormal and frustrating environment, fraught with all sorts of worries, fears, and uncertainties not ordinarily encountered in the same proportions in civilian life. Most individuals can make the adjustment, but these people cannot. In order to dispose of them their basic difficulties had to be determined, and an appropriate diagnosis had to be made. These men had made a satisfactory adjustment to civilian life. They were only disabled *so long as they remained in a military environment*. During the latter part of the war, the Navy gave official recognition to the fact that these men were not disabled for civilian life, by introducing the diagnosis "No Disease - temperamentally unsuited for military service" which came into very wide use.

Having pointed out that a very large proportion fall into the above category, let us return to the group as a whole. We must not forget that many serious mental disturbances were brought to light and diagnosed during military service. These ranged through the increasingly severe degrees of psychoneurosis to the actual psychoses.

There was also a special group not seen in civilian life, the Combat Fatigues and Operational Fatigues precipitated only by unusually stressful conditions. The important things to remember are that these conditions required most unusual degrees of stress to bring them about, and in their pure form they were temporary in nature. It is important to point out

that "War Neurosis" is quite a different thing. The symptoms were qualitatively the same but quantitatively quite different. The usual recovery did not occur following rest and removal from the precipitating environment. This diagnosis implies a rather severe psychoneurosis induced under stressful military situations in an individual who was probably not very stable in the first place.

We are now interested in appraising the entire group who received a psychiatric discharge as good or bad risks for life, accident, or health insurance. In endeavoring to pick the sheep from the goats we have five sources of information as to the nature and severity of their disabilities.

- 1—The Applicant's Story.
- 2—The Agent's Comments
- 3—The Medical Examiner's Report
- 4—The Service Health Record
- 5—The Inspection Report

There are really six but I have deliberately left out the Veterans' Administration because of the practical difficulties encountered in obtaining information from this source under present day conditions.

Let us now examine the potentialities and shortcomings of these various sources of information.

The Applicant's Story

It is only part of human nature to put one's best foot forward. The applicant is satisfied that he isn't *crazy*, and chooses to believe that this is all that should concern us. He has a firm conviction that all psychiatric diagnoses carry at least an implication of some degree of insanity, and accordingly feels free to disagree with the diagnosis, and puts his own interpretation on the story. He feels that this history is his own business. It is not very flattering. He is not proud of it, and he doesn't feel obliged to give it to you.

His idea that a neuropsychiatric history is his own business has been given support by various official measures, such as the omission of the diagnosis from discharge certificates, presumably in an effort to protect him from discrimination when seeking employment, and incidentally, insurance. As a matter of fact, we know that these well intentioned efforts in his behalf frequently boomerang by raising the very natural questions "Why all the secrecy?" "What is this history that is so jealously guarded?" "How much more is there to this story?" The applicant nearly always plays down the history, and often puts forth some rather fantastic explanation in an effort to minimize such a history. In most cases therefore, his story must be regarded rather critically, especially if there seem to be inconsistencies.

The diagnosis alone when volunteered by the applicant is deceptive. A diagnosis gives you no idea whatever as to the severity of a psychiatric condition. "Psychoneurosis" by itself can imply anything from mildly compulsive and perfectionistic tendencies, which in certain walks of life are a decided asset, to severe conditions approaching psychotic proportions. You cannot even say, "Well, it must have been pretty severe to necessitate a discharge." It may have been only one of several factors making discharge advisable. Unfortunately medical discharges are like some death certificates in that only one diagnosis is permissible. A marked degree of hypertension or even a coronary history may lurk in the background behind a diagnosis of some fairly innocuous psychiatric condition.

As physicians we realize that there is room for honest difference of opinion in psychiatric diagnosis. One man is impressed by one aspect of the case, while another sees the problem in a slightly different light. In addition, psychiatric diagnoses often do not represent clear cut entities which are uniformly interpreted and understood by all. In fact a good psychiatrist will often try to impress upon his medical colleague the fact that a neuropsychiatric diagnosis is seldom entirely satisfactory, and is of relatively little importance

anyway. *The clinical picture and history, as a whole, must be the primary consideration.*

The Agent's Comments

The agent is an interested party. He will endeavor to put a good light on his case, even though he knows that his client was tagged with a psychiatric diagnosis while in service. If his applicant is now working and looks well, he will consider him to be a first class risk.

We cannot look to the agent for any particularly useful information in appraising this type of risk.

The Medical Examiner's Report

Though few of our examiners are psychiatrically trained, and often they have only the benefit of a few minutes observation of the individual while completing the medical examination, they can give us a very useful appraisal of the risk, and I believe that considerable weight should be attached to the examiner's impressions regarding the significance of a psychiatric discharge. This is particularly true if the examiner happens to have been in military service himself and understands the mechanics of military discharges.

When necessary we would do well to go back to our examiner, present him with the known background, (which may not have been admitted to him), and ask him for his impression of the risk. When properly questioned, an applicant will usually give an honest, reasonable and convincing account of his history which, taken in conjunction with the examiner's personal opinion, may well permit of the acceptance of the case. If the examiner happens to know the man and his background, so much the better.

The Service Health Record

This is a folder which accompanies a man's unit throughout his entire military career, from induction to discharge. In it are entered the results of all routine physical examinations,

resumes of his illnesses and hospital admissions, copies of reports of medical survey, the recommended and actual disposition made, be it "full duty", "limited duty", or "discharge", as well as other miscellaneous information of a medical nature.

The Service Health Record is probably the best source of factual information on which to base an opinion as to a veteran's insurability. Nevertheless it has its shortcomings. The first of these is that in these psychiatric cases you often can't get it. You can't get it because of the applicant's attitude toward such a history and his reluctance to give it to you. You sometimes can't get it because of a law prohibiting the release of information which might prove injurious to the physical or mental health of a veteran. This law, as a practical matter, applies only to neuropsychiatric cases.

When you do obtain a copy of the Service Health Record there is some reading between the lines to be done. You must remember that the official disposition of a military misfit was often tossed into the lap of the psychiatrist. He must say enough to justify his recommendation that the man be discharged. At the same time he must avoid a report which will, on paper, make the man eligible for an unwarranted amount of pension, thereby handing him a crutch and performing that easiest of all tasks on earth, convincing him that he ought to receive a sizeable pension for the rest of his life.

In reading reports of medical survey you must remember that many were ready to use a degree of instability for purposes of discharge and possible pension, and some of these reports must be considered to have much the same relation to life underwriting as does a claim report of an accident. You are all familiar with the multiple abrasions, contusions, concussions, subluxations, and ecchymoses of the claim report which are miraculously converted into "a few scratches and shaken up a bit" when applying for life insurance a few months later. In the same way a middle course has to be steered in interpreting Service Health Records.

I have dwelt at some length on the shortcomings of these sources of information. I have not done so with a view to making the problem appear complex to the uninitiated, nor in a negative frame of mind—in fact quite the contrary.

In attempting to arrive at a practical solution to this problem it seems to me that all of the sources of information can be important, and occasionally all of them must be tapped in order to intelligently appraise an individual case. However, each must be properly evaluated and considered in conjunction with the others.

I now come to what I consider to be the most important point of what I have to say to you this afternoon, and that is, that in my opinion, the acceptability of veterans discharged with a neuropsychiatric diagnosis rests chiefly on the sort of an adjustment they make to a civilian life. Our rate structure has provided for individuals with mild degrees of nervous instability in the past. We have no desire to super-select today. We do not wish to penalize any individual who has been put over an unusually high set of hurdles and has tripped up. You must ask yourself "How did the applicant get along prior to service?" and "How has he made out since?" Some of you will say—this is well and good but how are we to avoid overlooking an occasional individual with a history of a genuine psychotic episode. There is a tip-off, if you can develop it. Psychotics were always discharged, and they were always discharged to a Veterans' Administration or Public Health Service Hospital. True, they may not have stayed there long, but they were not released to their own custody directly from the services.

It seems to me that the inspection report could be a most fruitful source of the special information we require to determine a veteran's civilian adjustment and insurability. For this particular purpose, present day routine reports are practically useless. We know that our various inspection companies have been plagued by man power shortages, and have lost experienced help. My purpose is not to criticize them but

to offer what I hope will be taken as constructive suggestions with a view to giving us better information on which to underwrite with greater accuracy the application of the discharged service man.

Most veterans have now been out of service for about a year, and it is becoming possible to determine what sort of civilian adjustment they are making. If the inspection companies can cover the desired information, their reports would serve well as a warning flag, as a check on other information, and as a real aid in determining the significance of a psychiatric history.

To be more effective, an inspection report on a discharged veteran should put greater emphasis on the securing of accurate information on the date of discharge, reason for discharge, and in the case of a medical discharge—whether he receives a pension. It would be most helpful to have the answers to the following questions: Is he currently receiving treatment? How long did he serve? Was he in the tropics? Did he have prolonged periods of hospitalization? Was he ever a patient in a Veterans' Administration or Public Health Service Hospital? What was his work record prior to service? How has he readjusted to civilian life? Did he resume a civilian occupation shortly after discharge? Did he return to his former occupation? Has he changed jobs since his discharge?

While I do not suggest that such an inspection will cover all situations, I believe that in most cases such a report in conjunction with our medical examiner's findings and diagnosis would permit of waiving official service health records in many cases, the securing of which, as we all know, results in delays and annoyance to all concerned.

PRESIDENT STREIGHT—Here is a very interesting subject. We have many in our membership present who have served in the various theatres of the war and who probably have opinions to offer. We would be glad to hear from you.

DR. ARTHUR E. PARKS—We ran into a fair amount of enteritis in the Army, and recently in Canada. It would appear that the problem is getting to be more important than it has been in our D V A Hospital, which corresponds to your Veterans' Administration. We find that more and more cases of enteritis are appearing. In the recurrence of dysentery, in fact, it appears that cross-infections are occurring in returned veterans and the wives and children of these said veterans are involved in enteritis. Further than that, it is becoming a problem that is very hard to cure and various new forms of treatment are being devised.

I feel that we should have that subject very much under consideration, and it should be regarded as a serious public health problem, because it is known that the ordinary methods of sterilization by water may fail in this particular disease. I would like to leave that thought with you, that in the case of amebic dysentery we should have a fairly serious attitude toward those cases, being very sure that a reasonable period of time has elapsed for the apparent cure of cases.

DR. ALBERT L. LARSON—I would like to agree wholeheartedly with what has been said about hepatitis. There is no distinction between hepatitis and jaundice. The only thing is we saw a great number of them in the war, and I could say the same thing about outbreaks in asylums, and all around. As far as Army routine was concerned, these cases were very carefully studied and watched. As far as all the available practical liver function tests were concerned, these were done on these cases before they were allowed out of the hospital. From our point of view then, I can say it doesn't present any underwriting problem.

DR. JOSEPH W. JOHNSON—I appreciate Dr. Hutchinson's remarks and Dr. Wilson's remarks and Dr. Parks' remarks. During the war I had an opportunity to serve as a psychiatrist, first, in the induction centers and, second, as Chief of the Psychiatry Service in Italy for the next two years, and they

made a work record prior to service and a work record after service. So far as I can see, the only practical way or means we have to appraise this extraordinary problem, the only way we could take these men, men who never had a job until they came out of the service, is to appraise them on the basis of how they handled situations in school, whether they had any reserve for the toughness of military service. We saw a great many men that we have passed into service as pretty good risks. We were appraising risks on the induction board, but very frequently we had to do a sloppy game of poker. Is this man a good risk for military service? Well, you saw those men break in combat; you saw excellent men break in combat in Italy where I was for two years. I was a member of the 34th Division. Months and months later these men would go on into a very serious type of depression.

The only means I can see of practical use in appraising them is the point Dr. Wilson made: Can those men make adjustments to civilian life—not only neurotic men but psychotic men—immediately after being discharged from the war? There were a few psychotic breaks in combat and I sent them home. But on their way home they would be stopped, for instance, in Africa, and they would make a reasonably satisfactory combat adjustment, and the men who had shock treatment and either electrical therapy or insulin therapy were sometimes returned to active and useful military service.

I don't know how we are going to appraise those men when we get a report from the Veterans' Administration that they have had shock treatment. I think we have got to look at all these people, those who have had psychotic breaks, and in the future I do not think we can assume they will not have psychotic breaks again. I am not as sanguine as many of my colleagues about that. Those men are not now fundamentally psychotic problems because they think they have security in this country, but later, in a number of years, we are going to have disability problems in these cases. They are going to crop up. Certainly from the practical appraising

viewpoint there can be nothing as useful as what Dr. Wilson and Dr. Hutchinson suggest—Can they work and are they able to begin work soon after getting out of military service? I wish we had a more definite means of appraisal but I don't know what it is.

DR. ARTHUR J. MCGANITY—I would like to ask Dr. Hutchinson if there has been any arrangement made in the American services for the review of these repatriated prisoners of war. I understand—and I speak subject to correction—at the present time we are in the process of reviewing all our repatriated prisoners and have been finding a certain very large number who have developed disability since they were discharged, or since demobilization and repatriation, through tuberculosis and some other diseases. I would like to know if there has been any attempt to survey such a group in the United States or not.

PRESIDENT STREIGHT—Are there any other questions anyone would like to ask? Do you care to add something, Dr. Hutchinson?

DR. HUTCHINSON—First, as to hepatitis, I have just within the last three weeks two death claims in my company that died as a result of acute hepatitis. Dr. Stokes, Jr. of Philadelphia, and Dr. Janeway, pathologist of Boston, have been studying this problem and I think something will break on this before very long, through conversations I have had with them directly and indirectly as to the possibility of hearing something more about hepatitis.

As far as a special program for the review of the prisoners of war, I am sorry but I know of none at the present time. I think that there maybe. I think it would certainly be a step in the right direction to have a review of some of these cases I have seen that have come through our company. They don't look too good, even after they have been back home a year or two and under proper nourishment and treatment.

PRESIDENT STREIGHT—The next speaker is Dr. Francis R. Dieuaide who spoke to us yesterday in his capacity as Scientific Director of the Life Insurance Medical Research Fund. He will now tell us of his "Wartime Experiences with Tropical Diseases and Their Future Significance." When war was declared Dr. Dieuaide was Clinical Professor of Medicine at the Harvard Medical School and Visiting Physician at the Massachusetts General Hospital. On leave of absence from this post he served as Lieutenant Colonel and Colonel in the Medical Department of the Army. He was assigned as Chief of the Tropical Disease Treatment Branch on the staff of the Surgeon General and was awarded the Legion of Merit for his services. Dr. Dieuaide!

DR. DIEUAIDE—I must apologize for speaking a second time before you, but it has been thought that you might be interested to hear a brief summary of experience with so-called tropical diseases during the war and such ideas as I can give you of the future significance of this experience . . .

WARTIME EXPERIENCES WITH TROPICAL DISEASES AND THEIR FUTURE SIGNIFICANCE

By FRANCIS R. DIEUAIDE, M. D.

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INTRODUCTION

Some years must elapse before a detailed, accurate history can be written of the experience of the Armed Forces with tropical diseases during World War II, or the final evaluation can be made of that experience. My purpose is to present to you a concise survey of this subject in the light of information available today. The data used pertain to the experience of the Army, but the experience of the Navy was essentially similar. It is a satisfaction to state that the two Services worked with uniformly close co-operation in the management of tropical diseases.

Many important diseases peculiar to the tropics can be prevented by the use of appropriate methods, some very thoroughly, others less satisfactorily. The Armed Services endeavored to make the utmost use of available preventive measures and in general their endeavors were highly successful. There were times in the course of military operations, however, when important preventive measures could not be efficiently applied. Furthermore, no such procedures were available in effective form against some of the tropical diseases to which our Forces were exposed. It happened, therefore, that a sizable body of soldiers and sailors acquired one or another of the diseases peculiar to the tropics. Every effort was made to provide for these patients the best therapeutic management that the medical profession could offer.

Parallel with this practical experience of the Armed Forces, the most intensive and elaborate research was undertaken for the improvement of both preventive and therapeutic manage-

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ment. These investigations were pursued almost entirely under the direction and with the support of the Office of Scientific Research and Development, but the Armed Forces endeavored to do their share of research also.

MALARIA, DENGUE, AND SANDFLY FEVER

As is known to all, malaria created more difficulties than any other tropical disease. During the war, the Army had nearly 450,000 hospital admissions for malaria.* Several whole divisions were put out of action and kept ineffective for long periods because of this disease alone. It was found that two-thirds to four-fifths of the patients relapsed, often many times over, in spite of the best available treatment. At one time in 1943 it was thought that malaria might render impossible our projected military program in the Southwest Pacific. There are three good reasons for this untoward experience. The first lies in the difficulties met in applying preventive measures, without adequate preparation, in strange environments, and in the face of the enemy. The second is the sad truth that no available medication was capable of radically curing the vivax infections which caused the trouble. Quinine was found to be a relatively inefficient and troublesome drug. The third explanation is found in the non-immune state of practically all our personnel and in the marked relapsing properties of the vivax strains of the Southwest Pacific.

I might inject a remark about quinine, that it is a drug which all of us feel we know something about. Whether we actually know much about it or not we have all learned something in the past about it. Some of us may very occasionally make use of it. A few of the medical profession, as a whole, in the United States make more than occasional use of it in the treatment of patients in those parts of the country where malaria actually exists. However, it is noteworthy how strong our traditions are. The tradition in favor of a drug, which we feel we know, persists long after the evidence that it is noth-

*Not including the Bataan experience

ing like as good as we have thought or that better drugs have been evolved and are available.

It is also true that there has been, in the case of malaria, at any rate, a remarkable prejudice to certain drugs, in spite of overwhelming evidence in their favor.

There is another side to the picture. Although patients were not permanently cured by treatment, malarial attacks were terminated with a speed and thoroughness which progressively increased as time went on. Improved knowledge of the use of atabrine made it possible to relieve all symptoms and remove all parasitemia within 48 hours in most cases. Only 226 deaths in all were attributed to malaria,* and some of these were due to other causes. By the summer of 1944, the situation had been brought under complete control as a result of improvements in preventive measures and in their application and as a result of advances in knowledge of how to use constant small doses of atabrine as a suppressive agent. As time went on, it became clear that, even though atabrine suppression was discontinued, more and more of those infected with malaria ceased to have relapses (provided they were not reinfected). This must now be attributed to the development of immunity.

In the meantime, research led to vastly improved insect repellents and to the development of DDT, which came to play a vital role in the control of many other diseases as well as malaria. Research, in the course of which well over 10,000 drugs were examined, also led to a new anti-malarial drug, chloroquine, which is even more powerful than atabrine (so powerful that two tablets taken once a week will thoroughly suppress malaria). Even this new agent, however, does not produce radical cures of vivax malaria. Late in the war, one of the new drugs, pentaquine, was found to be capable of radical cures, but the development of our knowledge of it, which is not yet complete, came too late for practical war-time application.

*Not including the Bataan experience

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It has been thought that the importation of malaria by returned personnel might cause a very serious increase in malaria in this country. The effect of this experience is not yet completely known. So far, however, we have passed through two summers (1945 and 1946) without any change in the incidence of malaria which can be attributed to returned Service men. Experts have predicted secondary cases of malaria derived from this source, but only in limited areas and only for a few years. The factors which have been responsible for the great decline in malaria in this country will continue to protect us.

It should be noted that during the war many parts of the program for the protection of the country against malaria have been improved and intensified, largely as a result of some fear that there might be imported malaria, and, in a practical sense, as a result of there being more money being made available for anti-malarial campaigns. I might also state that anyone who is greatly interested in these so-called tropical diseases felt embarrassed to explain our careless attitude toward the disease. That is, in the country as a whole most of us are quite unaware of how much malaria exists in certain parts of the country and of the inadequacy of the measures finally to suppress the disease or even to approach eradicating it in this country.

Along with malaria, there were many cases of dengue (also a mosquito-borne disease) and sandfly fever. These diseases were a military handicap on occasion, but they are non-fatal, brief, self-limited infections. They were not introduced into the United States during the war.

LEISHMANIASIS

This group of protozoan infections is mentioned because it is entirely foreign to the United States. Some 800 soldiers acquired the Middle Eastern purely cutaneous form of the disease, which is a chronic mild infection. A very small group,

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about 60 in all, were infected with the visceral type or kala azar, a more serious disease. Fortunately, efficient chemotherapeutic agents are available in organic antimony compounds, of which neostibosan is the best. These soldiers have probably all been cured. No secondary cases have arisen and the danger of implantation here is believed to be negligible, since it is doubtful that the necessary sandfly vectors exist.

RICKETTSIAL DISEASES AND RELAPSING FEVER

Scrub typhus, or tsutsugamushi, a rickettsial disease spread by certain species of mites, from the point of view of the patient was a far more serious disease than malaria. There were nearly 7,000 cases acquired in the South Pacific, with a fatality rate in some groups as high as 25 per cent. Early in the war, little was known about the prevention of this infection, but in time sanitary measures were evolved which proved most helpful. The disease was not imported into the United States and is unlikely to be because of the specific nature of the mite vector.

Until recently, no effective chemotherapy was known for scrub typhus or for any of the other rickettsial infections. In the latter part of the war, however, an agent was found which gives promise of efficacy in the whole group of diseases and especially in the mite-borne variety. This chemical is para-aminobenzoic acid, a substance of special interest in both bacteriology and chemotherapy.

Other forms of rickettsial disease were unimportant in our Forces. There were some 400 cases of flea-borne typhus and about 60 cases of Rocky Mountain spotted fever, all in this country. Louse-borne typhus, the age-old scourge of armies and war-torn peoples, was reported in 61 instances. This disease is rarely a problem under any circumstances in the tropics. In Europe, its control is largely attributable to DDT.

The status of immunization against the rickettsiae is still somewhat obscure, although the procedure is clearly helpful against louse-borne typhus and Rocky Mountain spotted fever.

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Nevertheless, with immunization available and with the modern sanitary procedures at our disposal, we possess the means of controlling this group of diseases. If these preventive measures are adequately applied, we need not fear the rickettsial diseases in this country. The war has only improved our knowledge of their management.

Relapsing fever, also an historical wartime affliction, often associated with typhus, was reported in about 240 cases.

HELMINTHIC INFECTIONS

Bancroft's type of filariasis was contracted by men of both the Army and the Navy who were stationed on certain South Pacific Islands. Some 2,100 cases were reported in the Army. In a certain percentage of native cases, as is well known, filariasis causes a disastrous end-result in the shape of the deformity known as elephantiasis.

I might say, by way of explanation, that one should not use filariasis and elephantiasis as synonymous. They are not at all. Filariasis is a specific disease which sometimes becomes associated with a pathologic change characterized by swelling in parts of the body, which is called elephantiasis. Elephantiasis may be caused by many other means than this infection. Since soldiers and sailors who had contracted, or who thought they had contracted, filariasis saw natives with elephantiasis, a troublesome psychological problem was created. Unfortunately, no effective chemotherapeutic agent was known and research started since the war has not yet been successful in finding one. Much valuable new knowledge of the early symptoms of filariasis was gained from this experience.

Investigation showed that elephantiasis is relatively rare among natives even where filariasis is very common. It turned out that the early symptoms of the disease were transient in the great majority of cases. Only in rare instances did any sign of the disease persist. It is believed that the infections acquired by American personnel were so light that few if any will develop elephantiasis.

Filariasis is known to have existed in South Carolina in the past, but new cases have not been reported for many years. The disease appears to have died out. Experience has shown that filariasis is not easily implanted where it is not naturally found. For example, in spite of the importation of infected workers from various Caribbean islands, the disease has never been established in Panama. As a matter of fact, parasitemia (which would be necessary before mosquitoes could transmit the infection) was rarely present in military or naval patients. It seems very unlikely, therefore, that filariasis will gain a foothold here as a result of wartime overseas infections.

Schistosomiasis japonica is one of a group of diseases caused by a class of flatworms known as flukes. The parasite in this instance has a complicated life cycle in the course of which it must live in certain specific snails. Man acquires the infection through bathing or washing with infected water. The Oriental form of schistosomiasis involves particularly the intestinal wall and the liver. In a certain percentage of natives who are infected over and over again in the course of years, the disease results in a crippling end-picture which is indistinguishable from Banti's syndrome. This disease is common on some of the Philippine Islands. Although warnings were issued, soldiers at first found it hard to believe that perfectly clear, clean water could be dangerous if it was not taken internally. As a result, in the early weeks of the Philippine campaign some 2,000 or so infections occurred with *Schistosoma japonicum*. Treatment of this disease is difficult, although certain antimony compounds are helpful (the best to date is the old-fashioned tartar emetic). Great care was used to treat all patients thoroughly. In a very small group of cases, perhaps a dozen or so, severe disturbances were produced by the accidental passage of eggs of the parasite into the brain. In most of these instances, the patients ultimately recovered; and all of the rest are now well as far as is known.

As in the case of filariasis, these schistosomiasis infections, which were acquired often at a single exposure, in all prob-

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ability were relatively light in comparison with those which result in severe permanent damage in native patients. It is not expected that such damage will develop in the years to come in more than a very few of the soldiers who were infected in the war. To date, in spite of a diligent search, no American snail has been found to be capable of harboring the parasite, so that the risk of its becoming established here is considered small.

Infections with the so-called old-world hookworm, *Ancylostoma duodenale*, were very common during the war, at least in the Army. This parasite, although in a general way similar, is more troublesome than the hookworm which is common in some parts of the United States. In particular, it is somewhat more difficult to eradicate. Some concern exists, therefore, over the possibility that this foreign species may establish itself in this country. Only time will tell whether natural forces, combined with the vigorous efforts which were made to eradicate the infections, will prevent such an undesirable event.

Hookworm infections are often asymptomatic and are rarely serious for the individual who receives adequate treatment, such as was given in the Armed Forces. It is unlikely that the health of any of the military patients suffered in any significant degree.

ENTERIC INFECTIONS

Infections of the intestinal tract occur entirely because of inadequate sanitary precautions in the handling of food and drink. In time of war, when haste, crowding, and confusion inevitably exist on some occasions, these infections are all too likely to spread. In World War II, the reliability of immunization against typhoid fever was proved anew. The incidence of the disease was negligible, as it was in American troops in the first World War.

Immunization against cholera is short-lived and probably not as reliable as that against typhoid fever. Fortunately, even though troops were stationed in significant numbers in

India and China, where cholera was not uncommon, sanitary measures protected our personnel from the infection.

No available measures of immunization are effective against bacillary dysentery or amebiasis. In some areas of military operation, including New Guinea, India, and North Africa, there were periods when sanitary precautions and discipline were inadequate. As a result, the number of dysenteric infections was considerable. Sulfonamide therapy was highly satisfactory in bacillary dysentery. Of the various drugs, sulfadiazine is the best in most cases and sulfaguanidine is decidedly inferior. The treatment of amebiasis on the whole is thoroughly unsatisfactory, since many drugs often must be used repeatedly. Nevertheless, in the majority of cases, the patients were ultimately cured. It is significant and fortunate that the establishment of chronic infections, either bacillary or amebic, which respond badly to treatment, was a rare event. It is believed that relatively few carriers of either infection were returned to civil life.

Apart from the satisfactory development of sulfonamide therapy for bacillary dysentery, there were no important new developments in the treatment of the enteric infections.

SKIN DISEASES

The circumstances of military service in wartime were associated with a high incidence of diseases of the skin. This occurred in all areas, even in the United States, but cutaneous ailments were especially numerous in tropical areas. These disorders arose from numerous varied causes and manifested themselves in many guises. In the tropics, the more important etiological factors in general were excessive perspiration, poor hygienic care of the skin, inadequate protection of the body surface (especially that of the arms and legs), and numerous small traumata which were often not properly treated. Important groups of patients with skin diseases resulted from two special causes, namely, sensitization in the course of the prolonged use of atabrine, especially in high dosage, and in-

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fection of skin lesions with the diphtheria bacillus. Unfortunately, overtreatment of skin diseases at the hands of medical officers with inadequate experience in this field often aggravated the basic ailment.

Skin troubles due to the prolonged use of atabrine attracted much attention because of the somewhat special nature of the lesions and because of the large numbers of cases which were determined by the fact that hundreds of thousands of individuals took atabrine routinely. Sensitization to many other drugs, especially the sulfonamides, also was troublesome.

Cutaneous diphtheritic ulcers are practically unknown in modern civilian practice. They result from the chance infection of small traumatic lesions which is favored by bad hygiene and overcrowding. Infection spreads mainly from carriers, either among the troops themselves or among the natives (who, contrary to previous impressions, not infrequently harbor the diphtheria bacillus).

And somewhat to our surprise it was proved that diphtheria is not a rare infection of the tropics. The natives of the tropics are as frequently carriers of diphtheria as the people of Northern areas. It is possible that a rise in diphtheria, which was rather marked in this country last autumn, last winter and spring, of this year, may have in part been caused by returning soldiers.

The names commonly used for skin disorders among soldiers, and even by some doctors, afford no clue to the nature of the disease in question. Among these were "tropical ulcer," "jungle rot," and "New Guinea crud."

Skin diseases rarely had fatal issues, most of which were due to explosive generalized exfoliation. Although prolonged incapacitation occurred in some cases, these diseases on the whole responded well to proper treatment (for which, however, return to this country often appeared to be necessary).

HEAT EFFECTS

It was expected that the effects of exposure to heat in tropical areas would be serious. For this reason, many organizations went through strenuous training under semi-tropical conditions in this country. In actual operations overseas, however, the effects of the high temperatures of the tropics, at least those of an acute nature, were much less grave than had been anticipated. Slightly under 200 deaths were attributed to the direct effects of heat on soldiers, but two-thirds of these fatalities occurred in this country.

DISCUSSION

It will be noted that no reference has been made to several tropical diseases of great importance. The reason is that these diseases either did not occur at all in our Armed Forces during the war or occurred only in a minute, entirely negligible, number of instances. Among such diseases, reference has indeed already been made to cholera and louse-borne typhus fever; others are plague (both bubonic and pneumonic), leprosy, melioidosis, Oroya fever, trench fever, yellow fever, trypanosomiasis (African sleeping sickness), leptospirosis (spirochaetal jaundice), and yaws. It is also to be noted that poisonous snakes, sharks, and other potentially dangerous animals had no significant effect on the health or activities of the Forces.

When the total effect of disease in the tropics is considered, it is obvious that the sum constituted a serious handicap to the Armed Forces, at least in terms of incapacitation of personnel. It must be remembered that venereal and other diseases affected the troops in tropical areas, as well as so-called tropical diseases. The war in the Pacific areas was often called a war of diseases, in contrast to that in Europe, which from a medical point of view was largely a surgical war.

Nevertheless, the part played by tropical diseases, even in the Pacific, should not be exaggerated. If we recall that fifteen million individuals saw some military service and that

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about three million served in tropical areas, we see that with the exception of the numbers given in connection with malaria, no figures here mentioned for any single disease have any statistical importance whatever. This is strikingly true when one considers the number of deaths due to tropical diseases, either individually or collectively. None of these numbers would appear on any chart of reasonable compass showing the main causes of death in the Service. Furthermore, as has been indicated seriatim above, it is unlikely that any but trifling numbers of individuals have been or will be permanently incapacitated because of tropical disease. As a matter of fact, accidents in the Services, to which surprisingly little attention has been paid by the public, caused far greater damage than disease.

On the other hand, the point of view of the individual should not be entirely neglected. Thousands of men and women suffered greatly and hundreds lost their lives because of tropical diseases.

In general, it is safe to say that the mere history of having had a so-called tropical disease in wartime service need not be regarded as a basis for refusing applications for insurance or for advancing premiums. This is especially true of a history of malaria. Only in case an examination shows some important pathological change or actual incapacitation would it be necessary to consider writing up a premium or refusing an application. Appropriate special examinations might be wise in cases in which there is a history of symptoms within the preceding year of amebiasis, kala azar, filariasis, or schistosomiasis.

It has also been indicated that there is so far no reason to believe that any exotic disease has been transplanted from tropical areas to the United States. It is likely that there have been and will yet be sporadic instances of certain infections contracted in this country which are traceable to returned service personnel. But it may be expected that the ultimate total will not be large and that such cases will cease to appear

before long. The dire predictions made early in the war were much too pessimistic.

From what has been said it is apparent that during the war our citizen Army and Navy achieved an excellent record in their struggle against tropical diseases. This splendid showing is due to high standards and loyal efforts in both preventive and clinical medicine. It is just cause for pride on the part of the medical profession of our country.

That tropical diseases did not take a greater toll in World War II, especially in the Far East, must be attributed in part to the relatively short duration of the war in densely populated areas, including the Philippines, China, Japan, and India. In these regions, except India, where only small numbers of American troops were ever stationed, operations went relatively well and fast for our cause. We did not have to meet the full potential forces in favor of diseases such as dysentery, cholera, and plague. The dangers of dense populations seeded with disease, combined with prolonged and untoward military operations in which inefficiency and confusion cannot well be avoided, are great indeed.

The advances made in tropical medicine during the war may be summarized briefly. Specific diagnostic and therapeutic advances were disappointingly few. At a very high price, some progress was made in the treatment of malaria. It is striking that penicillin, which has such a wide range of usefulness in other fields, was found to be efficacious in only a single tropical disease, namely, yaws. Streptomycin, which is still in process of development, has been shown to be very effective in tularemia, a disease often listed as tropical (though for no good reason); so far this drug has not given promise of efficacy in the treatment of any of the tropical diseases proper. Among preventive measures, the application of DDT and other means of insect control stand out for their success. The experience of World War II indicates that the condition of war is in no sense favorable to medical research.

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To have any chance of success, a frontal attack on a specific medical problem raised by warfare demands an enormous concentration of talent, labor, and money—an armamentarium far beyond that necessary in the more leisurely ways of peace.

Progress during the war in the knowledge of the course and general management of tropical diseases and in the understanding of the general significance of these diseases for the physician was perhaps more valuable than the specific advances which were achieved. In the past, tropical diseases have been considered to be a field apart from the rest of medicine. It is clear, however, that this group of diseases should be studied and managed in accordance with the same principles that underlie internal medicine in general.

We must look forward in the future to greatly expanded commerce of all sorts with the peoples of the Caribbean area, Central and South America, and the Far East. We must bear in mind the role which will be played by the airplane in this traffic and the speed with which the airplane travels. In these regions, so-called tropical diseases abound and sanitary conditions need enormous improvement. It behooves us, therefore, to devote in the future far more attention to research and teaching in tropical medicine. This is a most important lesson to be learned from the war. Only by adequate application to it can we prepare ourselves properly to protect the health of the United States, and to aid in the elevation of the standards of health in tropical regions with which we will have increasingly closer relations.

PRESIDENT STREIGHT—We are very much indebted, Dr. Dieuaide, for that splendid presentation of the significance of tropical diseases. The question is now open for discussion from the floor. Many of you have served in different areas. I am sure that you have a question to ask.

DR. E. CLARK NOBLE—I was very much interested in the remarks of Dr. Dieuaide in regard to the treatment of bacil-

lary dysentery. In my experience in the Air Force on the continent, shortly after VE-Day we encountered a great deal of dysentery, of which the etiology was not clear. I would like to point out that in the flying personnel who had the disease we noticed a marked loss of resistance. In that connection I think one or two lives were lost in operations where they continued to fly carrying several bomber loads per day. I think you know about the treatment of these Allied troop disorders, which I understand you saw in Italy, some of you. I think, perhaps, Dr. Parks saw that. He was largely with the Canadian forces as well as the American forces.

DR. CHARLES A. PETERS—I have enjoyed very much the paper of Dr. Dieuaide, and I don't understand how he covered quite so much in so short a time. I was a little bit disappointed that more wasn't said about amebic dysentery. I have been consultant to the returned soldiers in Montreal since World War I, and while it is true that malaria was a bigger problem in the war days, we find that dysentery is a much bigger problem in peace, and I am not so sure that in this country you are not going to have a good deal of amebic dysentery.

DR. ANDREW J. OBERLANDER—I would like to ask if you noticed any difference in the occurrence of malaria that you have seen in the Southwest Pacific and in the Mediterranean region. I had a little experience with the Marines when they first went to Guadalcanal. As you may appreciate, they had no suppressive treatment whatever. I might add another little word to our vocabulary which we see coming up on our applications and that is the word "mumu". If you are not acquainted with it, it is the common name that the Marines gave to filariasis. I agree that as far as my experience in the Navy is concerned, in the Pacific and in China, venereal disease is a problem in underwriting, and it is probably one of the most difficult diseases for diagnosis. Penil ulcers were very common. The appearance of typical ulcers was seldom seen, so the diagnosis of syphilis was made with difficulty.

Those men who spent any time out there at all were highly exposed to *plasmodium vivax*.

DR. D. SERGEANT PEPPER—I should like to ask Dr. Dieuaide about the revival of interest in plasmochin, which is a combination of quinine for suppressive or curative treatment for recurrent *plasmodium vivax* infections.

DR. DIEUAIDE—I am afraid I have already taken up too much time but the field is so enormous I would characterize my presentation as sketchy in the extreme. It would not have been possible for me to enter into the details and I deliberately avoided them.

As for the relative importance of dysentery, I certainly would agree, from a therapeutic point of view and from the point of view of the individual, it is as a whole, perhaps, more important than malaria. Certainly it can be called harder to deal with. That is especially true of amebic infections. I said, you will remember, that I regard the treatment of amebic infections as pretty thoroughly unsatisfactory.

With regard to the relative merits of the sulfanilamide drugs in the treatment of bacillary dysentery, to begin with, a great many patients with bacillary dysentery will recover clinically of their own natural forces, if we put them at rest and give them a proper dietary and fluid treatment. A most important consideration, however, is the persistence of the bacilli in the stools, which, although it doesn't go on for a length of time, is very common for a limited period of time, if the patient doesn't receive treatment.

The efficiency of the sulfa drugs has been in the disappearance of the organisms, I think, but I am a little in doubt. I put my emphasis on di-sulfanylguanidine for dysentery. The truth is that the efficacy apparently differs with a specific organism. I don't think the difference is important enough to pay much attention to it in the general practice of treating bacillary dysentery. Sulfanylguanidine interested us because it is highly concentrated in the lumen of the intestines and

works on the organisms there. However, that theory is a rather specious one anyway. The best way to reach those organisms is through the circulation. In fact, it is commonly believed now that the effect from sulfanylguanidine is probably due to the small proportion of that which is absorbed rather than that which is not absorbed. The use of sulfanylguanidine has to be carried out with greater care because of the relatively greater danger of intoxication of the patient. I believe that during the war a great many patients were treated under a lot of handicaps, and were frequently made to keep on duty when they should not have been, from a medical point of view, although it may or may not have been necessary from a military point of view. One has to distinguish sharply between the general ideal of general medical practice and that standard which one can achieve under the stress of military circumstances where actual operations are in progress.

As for the future occurrence of dysentery, specifically amebic dysentery in the United States, of course, we can't say. We won't know until we have some experience to go on, and I, as we say, stuck my neck out in saying that I didn't believe that it would be greatly increased. I don't doubt that among the individuals who acquired amebic dysentery during the war, a fair number may suffer continuously from amebic dysentery. I, perhaps, suggested more strongly than I should that I didn't think there were such patients. I do not mean to say that, but I think from the statistical point of view they would not turn out to have very much significance. It is a difficult matter, when you come to consider an individual with a history of dysentery for insurance. As I suggested here, one should certainly go into the case carefully, and I would propose that actual special examinations were in order unless the history was very vague and remote in time.

As to the differences of malarial infections between the Southwest Pacific and the Mediterranean, our evidence, which is fairly good, would lead to these answers: First, there is

no significant difference in *plasmodium vivax* as shown by the severity of the attack. Second, that there is some difference in regard to the relapsing properties of the strains and those properties are greater in the Southwest Pacific than in the Mediterranean. It has been proved conclusively that the Southwest Pacific strains that we had in soldiers are much more prone to relapse than those of the United States. Similar evidence exists in regard to amebic infections, but it is not as complete.

As to the use of plasmochin in the treatment of malaria, we carried out quite a thorough study of the usefulness of plasma, and we finally came to the conclusion that it is true, particularly in the patient who had malaria for some time and, therefore, has acquired some immunity, that relatively large doses of plasmochin given over a relatively long period of time are capable of curing some malarial *plasmodium vivax* infections. However, the circumstances under which you can safely give such treatment must include complete hospitalization for a period of about three weeks. This is a process which the Army could not use overseas, and we began to use it in this country early in 1945, but I would hesitate to recommend its use except by individuals who are especially interested in handling such problems. Plasmochin is a much more dangerous drug than most of the drugs we use in the practice of medicine, and it is particularly dangerous because from its effects there is sudden and catastrophic endomixis which occurs without any warning that we have been able to pick up. Very thorough investigations of patients have never revealed any warning which could be picked up to say that such a crisis was about to occur. Deaths in the Army due to malaria were probably fifty-fifty. I know, in about a hundred which have been studied histologically, it was found it was due to *plasmodium vivax* infections, but I also know an equal number that were not due to *plasmodium vivax* infections. That is, perhaps, a little surprising to those who haven't seen deaths from malaria occurring because of *plasmodium vivax* infec-

tions, but it is not surprising when the patient wasn't treated properly, and particularly if other elements than malaria are present, which is often the case in the Army.

The name "mumu" for filariasis was not made by the Marines. That's one thing they can't claim. It is the old and traditional name for the early symptoms of filariasis. It was the natives of the Southwest Pacific Islands from whom the Marines, in common with other service personnel, got the name of "mumu". It was from the natives with whom they often talked about the disease because of their fear of getting it themselves.

PRESIDENT STREIGHT—Dr. Anthony J. Lanza, Associate Medical Director of the Metropolitan Life Insurance Company will be our next speaker. Dr. Lanza has had a wide and varied experience on the foremost industrial diseases. Dr. Lanza!

INDUSTRIAL MEDICINE

BY ANTHONY J. LANZA, M. D.

ASSOCIATE MEDICAL DIRECTOR

Metropolitan Life Insurance Company

Industrial medicine embraces not only the emergency treatment of injuries occurring during the working hours and the subsequent care of compensation cases, but also health conservation and the control of working conditions insofar as their health aspects are concerned. Industrial medicine is the application of the principles of preventive medicine through the industrial approach.

An industrial medical program includes:

- a. Selection of the individual for the job which implies a preplacement physical examination.
- b. Periodic health examinations which are the basis of a diagnostic service for workers and the corner stone of health education.
- c. A close working contact between the plant medical department and community and other local health and welfare facilities.
- d. Supervision of eating facilities and a definite nutrition program.
- e. An adequate record system which is necessary for the effective administration of any health program.
- f. Strict adherence at all times to the standards of medical ethics.
- g. The control of occupational health hazards and the supervision of working conditions for the protection of the workers' health.

That sounds like quite a lot and it does include quite a range of activities, but their relationship to their intended purpose—maintenance of the workers' health—is logical and

practical. Such a program is in effect in many industrial establishments, and many more have a health program that may not be as complete as the foregoing, but will cover some of the more important features.

The return of war veterans has emphasized the necessity for proper selection and placement and it should be understood that no health and medical program can accomplish much unless preplacement physical examinations are carefully made and recorded. Unless the industrial physician knows the physical and mental condition of the individual worker, his efforts to maintain a medical service will be based on guesswork.

In reviewing the items above mentioned, it is obvious that one phase of an industrial health service, namely, the control of environmental conditions in a mine or factory or workshop of any sort, is an engineering rather than a medical function. We have developed, in this country, the industrial hygiene engineer who is concerned with illumination, ventilation, the control of dusts, fumes, gases, and noise. Mostly, but by no means entirely, the industrial hygiene engineer is concerned with atmospheric air—its movement, temperature, moisture content, and the various substances, originating in industrial processes, that may pollute it and so constitute a menace to health. The control of air and its content is to the industrial hygiene engineer what the control of water supply is to the sanitary engineer in a health department.

Without going further into this phase of industrial hygiene, it has been shown, especially in the war experience, that the industrial physician and the industrial hygiene engineer, working together, are a most potent force in doing sound preventive medicine. Alone either one can do much, but it is impossible to achieve an effective and satisfactory industrial health administration unless both specialists can work together.

The physician who serves industry either directly or indirectly and who is called upon to make diagnoses of industrial workers, should remember that there are 168 hours in a week, of this time seldom more than a fourth is spent on the job. Consequently, any one of us, regardless of the nature of our appointment, spends much more time away from work than at work. Occupational diseases, either acute or chronic, account for a very small portion of the time lost through illnesses by wage earners of all kinds, although the occupational disease picture may be extremely important in some industries and in some processes. We have to be on guard against two types of error arising from the failure of the physician, in many cases, to properly know and evaluate his patient's occupation. There is a snapshot diagnosis of an occupational disease based upon the fact that the individual may work in an industry which is supposed to have an occupational hazard. On the other hand, true occupational diseases are frequently missed because the physician has no conception of what his patient actually does. Both of these tendencies towards error plus imperfect or absent record systems and vital statistics, leaves us today with a very incomplete knowledge of the extent of occupational diseases. We know pretty well what types of industry and what processes or jobs in those industries can and do cause occupational illness. How much occupational illness actually results from these causes is still largely a matter of conjecture.

The extension of compensation coverage will, it is to be hoped, in the next decade or so build up more accurate figures on the incidence of occupational disease. In the meantime, those of us who are concerned with occupational rating have to be contented with what little information is available plus first hand experience and observation in endeavoring to be just towards those who seek insurance coverage.

We got some very striking examples of that during the last few years, but I will only mention one. Some time in 1944, I think it was, an alert industrial physician in New England

spotted a patient with some type of pulmonary involvement and he couldn't make a diagnosis, so he called in an expert to help. They began to look around and they found a number of these cases. To cut a long story short, these were people who worked in making fluorescent lamps—and then when they began to look around they found a few cases here and a few cases over there and a few cases down here, until now they have accumulated some forty odd of these cases with a mortality, I think, of approximately 50% so far and some of the remaining cases are not so hot.

Well, then you wonder how it was that so many of these people have been having this sort of thing and were never picked up—why they had never been diagnosed.

I have in mind the case of a young woman who became ill on a job and was sent to her family physician. He made a diagnosis of influenza and sent her to the local hospital, but at the local hospital they did a thorough job of examination. Among other things, they did the routine X-ray of the chest and when they did that, the cat was out of the bag. She didn't have influenza. Then they began to check up on her and they found that the year before she had worked in a fluorescent factory before she went to her present shop. That is the sort of thing that goes on and the only hope of ever getting at it, is just like what Doctor Pack was saying about the cancer cases. It's the doctor on the job that sees these cases that has to pick them up.

And our problems in that respect I think are going to become more difficult. Some time ago I was informed by Dr. Curtis, of the Bureau of Standards, in Washington, that coming out of the peculiar business of Oak Ridge, Tennessee, there were about two hundred industrial products that would be put into manufacture as soon as the patent and the legal red tape could be cut through so as to enable production to go ahead, and that these two hundred products, all of them, involved exposure to radiant energy in a highly dangerous degree. So on top of what we know now, we are faced with

an entirely new occupational hazard, about which we know only a little.

Now when they started work on the various plants that were concerned with making the atomic bomb, they set up a threshold limit for exposure. If I remember right, I think it was one-tenth of a radiant for an eight-hour shift, but the people who set up that standard acknowledged they had just hooked that out of the air, and based upon their experience they hoped it was all right, and so far everybody has got his fingers crossed, and we think it has worked, but with the extension of manufacture into many other substances involving radiant energy, it behooves us all to acquire as much knowledge of this subject as we can.

However, our knowledge of occupational hazards is increasing and the general interest that has been stimulated in industrial hygiene in the last few years implies that our efforts will be based upon a firmer foundation within the course of the next few years.

I would like to say just a few words more. The American Medical Association set up nearly ten years ago a Council on Industrial Health, and that Council is working through State and County Medical Societies and inducing the State Societies to set up a permanent committee on industrial health and inducing the County Medical Societies to set up similar Councils, particularly where they are in industrial areas, we hope, and are carrying on a campaign of education among the doctors with the hope of picking up more information so that when we have to, particularly in our business, determine whether the man's occupation calls for a higher rating or not we will have something to go on. That is true also of the Home Office Underwriters' Association, who, as you know, meet from time to time and consider this matter of occupational hazards, but too often the considering involves merely a rehashing of the literature, most of which goes back fifteen, twenty or twenty-five years and is entirely out of touch with modern industrial affairs.

There is one more thing that concerns all of us and that is the question of educational facilities in this particular field. Up to the present time the industrial physician has had to come up the hard way and he has acquired his knowledge of industry in its relationship to health by experience and by more or less the painful method of trial and error. At the present time it is a hopeful sign that at least a half dozen of our leading medical schools are trying to set up definite programs of instruction. Some of them are quite ambitious and include such a thing as an Institute of Industrial Medicine to teach both graduate and under-graduate students, and that program I suspect will begin to get under way pretty soon and ought to be effective within the next ten years. In the meantime, all the information that we can get on occupational hazards is little enough, and we keep on groping, but I think we are groping to more purpose at least than we were, say, ten years ago. Thank you.

PRESIDENT STREIGHT—This question is open for further discussion. Any questions? I am sure Dr. Lanza would be very glad to answer them.

DR. BERTHOLD R. COMEAU—Dr. Lanza mentioned the case of a young lady who had worked in a lamp plant. What was the nature of the work she did and what was the nature of the condition that was discovered?

DR. LANZA—The condition was a general pulmonary involvement that in some cases, as far as the X-ray film was concerned, resembles Boeck's sarcoid. It isn't Boeck's sarcoid. Just what it is, we do not know. The pathologic work has been done and is still being carried on at Saranac Lake. It is supposed—and this is purely an inference—that the active agent causing this is beryllium. There is some toxic action of beryllium which is an ingredient in the stuff they put in fluorescent lamps.

PRESIDENT STREIGHT—Our next speaker is Dr. Chester S. Keefer. Dr. Keefer is an M. S. from Bucknell University and received his M. D. from Johns Hopkins. He is the Wade Professor of Medicine at Boston University School of Medicine, Physician-in-Chief at the Massachusetts General Hospital and Chairman of the Committee on Chemotherapeutics of the National Research Council. He is going to speak to us about "Chemotherapy". This is going to be a very interesting subject for he knows all about it. Dr. Keefer!

CHEMOTHERAPY

BY CHESTER S. KEEFER, M.D.,

Professor of Medicine

Boston University, School of Medicine

In order to place these various chemotherapeutic agents in their proper perspective it may be well to review the subject briefly. I need not say to an audience of this kind that during the past ten years we have all witnessed surprisingly great advances in the treatment of infectious diseases. In fact, many diseases can now be treated adequately with a very small death rate, whereas when the same diseases were encountered 15 years ago there was no specific therapy. I need only recall the fact that pneumococcic pneumonia had an average mortality rate in most hospitals of between 20% and 30%—years ago. Today certainly the mortality rate is less than 5%. In fact, in one large camp in the United States, where Type 2 pneumonia was prevalent in two successive years, 1,500 consecutive cases were treated with sulfadiazine without a death. That is a remarkable achievement.

I think it is fair to say that the sulfonamides continue to be the drug of choice in the treatment of bacillary dysentery. There is little evidence that streptomycin influences this disease, but so few cases have been treated it is impossible to say what effect it will have on that disorder. During our studies we did not take up the treatment of dysentery with streptomycin, for the reason that the sulfonamides were so effective.

It should also be remembered that the sulfonamides are the most effective drugs we have for the treatment of meningococcic meningitis. It is true that penicillin will influence the course of that disease but the results are not nearly so impressive as is the case with sulfadiazine.

In certain cases of urinary tract infection the sulfonamides continue to be extremely effective and this may be true of cases in which the organisms have been found to be resistant to streptomycin. I shall return to this subject presently.

During the war when large numbers of men were being mobilized in various training camps, it was a common experience to observe outbreaks of hemolytic streptococcal infection of the throat. Sulfonamide prophylaxis proved to be extremely effective when the organism that was prevalent in that community was susceptible to the action of sulfadiazine.

Some of you may recall that several years ago most of the outbreaks that occurred in camps were due to a strain Type 19 hemolytic streptococcus, which proved to be highly resistant to the action of sulfadiazine. All of these infections, however, were susceptible to the use of penicillin.

At the present time there is a tremendous amount of penicillin being used in this country and abroad. In fact, the production in this country is said to be phenomenal. There are many problems that remain to be solved for the reason that the product that is used commercially is a mixture of penicillins. There are at least four penicillins which have been crystallized and studied by the chemists. They all have the same basic ring structure with different side chains. They are all biologically active. They are, perhaps, quantitatively different, as far as their activity is concerned.

No one has made any clinical studies of the crystalline product of each one of these penicillins known as F, G, K and X, so that we are in no position to say whether one crystalline product is better than another. Some experiments in experimental syphilis, suggest that crystalline K, or, at least, products containing not less than 90% K, haven't been as potent as crystalline penicillin G, but I should like to say there are no clinical studies in which comparative results are available to indicate whether one type of penicillin is more potent than another.

Until crystalline products of each penicillin become available and can be studied in a comparative way, we will not be in any position to say that one is any better than the other. Most of the products which are now available for clinical use contain mostly penicillin G. This is the product that is developed in submerged culture when chemical precursors are added to the media. When you buy commercial penicillin today it is mostly penicillin G.

One of the great difficulties that surround the discussion of penicillin has been that of dosage. That was inevitable, for the reason that we were dealing with a non-toxic drug. The maximum tolerated dose for penicillin has never been determined. I know of a young child three years of age who received as much as a hundred million units a day for a period of three weeks without showing any signs whatsoever of intoxication. When you are dealing with a non-toxic drug it is practically impossible to determine the maximum tolerated dose.

The same applies when you attempt to determine the minimum effective dose. When supplies are abundant, treatment is started for any infection. If the patient isn't well within 24 hours it is a common custom for the physician to double or treble the dose, with the hope that the disease may be cured or arrested within a very short period of time.

To give you a few specific examples, it is, perhaps, well to recall that many patients with gonococcal infection can be cured within 24 hours by using 150,000 units. Bacterial endocarditis, on the other hand, a much more serious disease, may require 300,000 to 500,000 units a day given for a period of three to six weeks; so that the time dose relationship will vary from one patient to another and from one disease to another. This must be taken into account when any assessment is attempted in the study of infections.

A great deal has been said, particularly in the lay press, from time to time about penicillin resistance. There are a few strains of staphylococci that have proved to be resistant

from the beginning, but in our own experience we have not encountered any resistant cases of hemolytic streptococcal infection. In our own clinic, under the direction of Dr. Fleming, we have not found any instances of penicillin resistance in gonorrhea when the question of reinfection has been controlled. A certain number of strains isolated from the circulating blood of patients with subacute bacterial endocarditis may be exceedingly resistant—but the vast majority of strains that cause infection of the heart valves are sensitive in amounts equivalent to less than a tenth of a unit per cubic centimeter. One comes to the conclusion that if strains of organisms are becoming penicillin resistant it is desirable to know how sensitive they were before treatment was started. Certainly we have seen nothing so far that would indicate that we have eliminated sensitive and susceptible strains.

Penicillin continues to be the drug of choice in the treatment of gram positive infections, and that includes the staphylococcus, streptococcus hemolyticus, the pneumococcus and the gram negative group of cocci.

We know that many patients with acute osteomyelitis will recover completely and remain well, without surgical treatment. Progressive healing of lesions in the bone occurs under treatment and continues after treatment has been discontinued.

We have also found that when penicillin is combined with good surgical treatment many patients who have had chronic osteomyelitis for years can be improved so that they have no signs of infection, and in many instances no recurrences over a period of three to four years. It is too early to say how many of these patients with chronic osteomyelitis will remain free from recurrences because it has been a universal experience that many of these patients have had recurrences in the past, as long as twenty to twenty-five years after the initial infection.

One of the most striking results that has been recorded has been that in bacterial endocarditis due to the non-hemolytic

streptococcus. We can say at the present time that the recovery rate, that is the recovery from the infection, is approximately 40%. A certain number of patients die early in the course of treatment, usually before they have been under treatment three weeks.

In many of these cases the treatment is started late in the course of the disease, and by late one means at a period when signs of cardiac insufficiency have already become established, or after cerebral accidents have occurred. Death in these early cases, that is, early from the point of view of treatment has been the result of an active infection, cardiac insufficiency, cerebral embolism or hemorrhage. A certain number of patients have a complete arrest of their infection and die within six months to two years after the treatment has been concluded. It is an interesting fact that the vast majority of these patients die of heart failure and not of reinfection. In our own experience, reinfection is most likely to occur within four weeks after the initial treatment with penicillin has been discontinued. In brief, you have a group of patients who have arrested their infection following one or more courses of penicillin. You have another group that dies before the initial course can be completed. There is a third group in which death occurs within six months or later. In the third group, death is due to heart failure or cerebral embolism and not as a rule to reinfection.

The best results have been obtained with a minimum period of treatment—of three weeks, preferably five, with at least 300,000 to 500,000 units a day.

In case of relapse, treatment is started once again and continued for a period that is longer than the initial treatment, and usually with larger amounts. Failures in our own experience have not been due to the development of penicillin resistant strains. Usually if the infecting strain is resistant, it is resistant at the very outset of the disease. A certain number of patients have recovered when as much as one to ten million units of penicillin have been given daily. In those

cases the strain has usually been resistant to as much as a half of a unit per cubic centimeter.

We have not been using heparin along with penicillin in the treatment of these patients, for the reason that the results obtained without heparin have been as effective as those when heparin was used. We can say that in most cases of known non-hemolytic streptococcal bacterial endocarditis, if treatment is started early and continued for a period of at least three to six weeks with amounts varying from 300,000 to 500,000 units per day, at least 60% will have an arrest of their disease. A certain number of patients with acute bacterial endocarditis also recover.

Patients with staphylococcus aureus, pneumococcus, and hemolytic streptococcal bacterial endocarditis have recovered in approximately 25% to 30% of the cases. These patients are left as a rule with signs of valvular defects, and while some of them remained well for as long as two years, others died within six months to two years, with cardiac insufficiency. The patients with pneumococcus and streptococcus infections who recover may then show signs of aortic insufficiency, have a period of well being from 6 months to a year and then develop symptoms and signs of heart failure, and disease a result of valvular defect and the mechanical disturbances which follow it.

I need not say anything about the use of penicillin in gonorrhea because it has such a profound effect that it has been widely publicized and is well known.

The studies that have been going forward in the treatment of syphilis indicate that we have a non-toxic drug, and that the results are certainly comparable to those which have been obtained from the use of the heavy metals. No less an authority than John Mahoney has made the statement within the last two or three weeks that we now have an agent which can be used effectively in the treatment of early syphilis. We have every reason to believe that the late manifestations of

syphilis can be abolished provided treatment is started early and continued to the point of effectiveness.

One can say that penicillin is still the drug of choice for the treatment of all gram positive infections.

We come now to another agent that has been under active investigation for a period of at least eighteen months, namely, streptomycin. Streptomycin, which is the product derived from the growth of certain strains of *streptomyces griseus* in a suitable medium, has a very profound effect in the test tube, at least, on a wide variety of gram negative bacilli. It is also able to inhibit and in some instances kill large numbers of gram positive cocci, but in much higher concentrations. When you study the various species of organisms, or even different strains within the species you find there is a wide range of susceptibility of various organisms to streptomycin.

This wide range of sensitivity of the various strains of gram negative bacteria explains in part, at least, the results that may be observed in the treatment of many infections. Of all the infections that have been treated in which streptomycin stands out as an extremely effective drug, tularemia heads the list. There is a sharp decrease in the temperature and constitutional symptoms within forty-eight to seventy-two hours. The fatality rate is greatly reduced and the total duration of the disease is enormously shortened. We now have records of approximately 125 cases and there have been only two deaths, and then only in patients who were treated for less than eight hours.

In the next group of infections that have been influenced favorably is meningitis. Here, when a combination of intramuscular and intrathecal therapy is used, the fatality rate can be decreased and the total duration of the disease shortened. A very interesting phenomenon has appeared during the treatment of some of these patients. We have found in several instances now that you may be able to eliminate the gram negative infection, and then be faced with a staphylococcal infec-

tion of the meninges or throat and lungs. It would seem that once you suppress one group of organisms with an antibiotic agent other organisms which are not susceptible to streptomycin grow and flourish and cause actual disease. It is in these cases that a careful bacterial study of secretions is very important in assessing the results of treatment during the disease.

The change in the character of the infection is important in the treatment of urinary tract infections. We have found, for example, that you may start with a mixed infection of the urinary tract with invasion of the blood by one of the organisms. The susceptible organisms may be eliminated. The existing ones flourish and may actually invade the tissue and cause infection due to an entirely different group of organisms.

It is a common experience to find nothing but the colon bacillus in the mouth and throat of patients who are using penicillin lozenges and troches, or patients who are using penicillin aerosols for the treatment of mixed infections of the lungs.

In urinary tract infections streptomycin is extremely effective. In about 50% of the cases, you will find that the urine can be sterilized at least temporarily and the clinical features of infection will disappear. In some instances bacteria persist or reappear after treatment is stopped, although there are no constitutional symptoms associated with it. In these cases, sulfonamides, or even nothing but a large fluid intake, may be responsible for a disappearance of the organisms from the urine. In our experience, when obstruction to the free flow of urine continues the sterilization of the urine is temporary.

The results in typhoid fever, following the use of streptomycin, have been disappointing. We have no evidence that it actually shortens the course of the disease.

With respect to undulant fever the same has been true. A number of patients improve so far as constitutional symptoms are concerned, with the use of streptomycin. So far we

have no evidence that the total duration of this disease has been shortened. From a number of physicians, who have had wide experience with this disease, especially those in Iowa, where the disease is likely to be an occupational one among workers in abattoirs, we have the statements and the records that the patients who have been treated have not relapsed, and it is the opinion of the physicians who have studied them that the convalescence has been shortened. In assessing this group of cases it should be remembered that in the experimental disease of the guinea pigs it has been necessary to continue treatment for at least four weeks before the tissues are sterilized. It is quite likely that more protracted treatment with streptomycin may actually shorten the total duration of many instances of undulant fever. Certainly the *in vitro* sensitivity of these organisms would suggest that it is a susceptible infection but the clinical results so far have been difficult to assess, for the reason that we have not followed these patients for a sufficiently long period of time.

What has been said about typhoid fever also applies to salmonella infections, or the paratyphoid group. Here we have very little evidence that streptomycin has greatly shortened the course of this disease. That is, perhaps, unfortunate, for the reason that most outbreaks of food poisoning that are encountered nowadays are due to salmonella infection and not due to dysentery.

One mixed infection with gram negative organisms predominating, in which the results with the use of streptomycin have been encouraging, has been in peritonitis. It has reduced the constitutional symptoms and in many instances has seemed to have contributed to the patient's recovery.

Every one is vitally interested in the results that might be anticipated from the use of streptomycin in the case of tuberculosis. I think it is fair to say that it is a highly effective agent in the treatment of experimental infection in guinea pigs, and while it does not sterilize the tissues in more than

70% of the animals, certainly the progress of the infection can be controlled.

In tuberculosis it is the reparative process that is usually slow. It is the acute process that tends to be rapid, so it may require treatment over a long period of time before any results can be obtained that will be assessed with any degree of accuracy. We certainly know from the work of Dr. Hinshaw and Dr. Feldman at the Mayo Clinic that many patients show an arrest of their disease and that there is no progression of the lesion while they are under treatment, but there is often a regression of the signs of exudation while they are under treatment. The treatment must be continued for, perhaps, at least three to six months, and perhaps longer, if the best results are to be obtained.

Obviously, too few patients have been treated and followed for too short a period of time to make any statement whatsoever concerning the outcome. I think that it should be remembered that in studying a disease like tuberculosis, the time factor is very important, whereas when you are dealing with pneumococcus pneumonia you think in terms of seven to fourteen days. In tuberculosis the minimum period is certainly five years and the maximum is a lifetime. One must face the fact that in tuberculosis you are dealing with a very chronic infection in contrast with acute infections. It would be best to treat and follow patients with tuberculosis for a longer period, than is necessary in an acute infection.

What are the reactions that you are likely to see following the use of large amounts of streptomycin? It is relatively non-toxic but it is not as innocuous as penicillin. Skin eruptions and fever turn up in 5% to 10% of patients. They are more common and more frequent when large amounts are given daily for a period of two to three weeks. One very troublesome symptom that has appeared in a certain number of patients has been vertigo. This is noticeable in ambulatory patients; it is much less noticeable in bed patients. It tends to regress after a period of several weeks and usually disappears

entirely. It has been stated by some groups who have studied this matter that if you do caloric tests, the abnormal reflexes persist. In another group, abnormal vestibular reflexes are present early but disappear entirely. Suffice it to say that this has not prevented long term treatment in patients in whom it has turned up. Certainly, it is not true, as one popular writer said a few weeks ago, "This man will be dizzy in the dark for the rest of his life." That, of course, is not so and has not been the experience of very careful observers.

None of these drugs that I have been talking about are effective in the treatment of virus infections, and that is one of the great fields of interest at the present time, for the reason that virus infections not only cause disability, but also in many instances they may be responsible for death. It is perhaps, not too much to hope that a new class of agents may be found that will be effective in the treatment of virus infections.

I should like to sum up this discussion by saying that we now have effective agents for the treatment of a wide variety of bacterial infections. The selection of the proper drug will depend largely upon knowing the bacteriological diagnosis and using the indicated antibiotic or other type of chemotherapeutic agent. So far none of these agents have been effective in virus diseases.

PRESIDENT STREIGHT—Thank you, Doctor, for your very splendid presentation. This paper is now open for discussion from the floor. I am sure there are many of you who would like to ask questions. Do not hesitate to do so. We have plenty of time and I am sure Dr. Keefer would like to answer them.

DR. ROBERT A. GOODELL—I am very much interested in Dr. Keefer's remarks—particularly his remarks this morning about bacillary dysentery and the effectiveness of sulfonamides. I was fortunate, or unfortunate enough, whichever way you want to look at it, to be stationed Chief of Medicine on a hos-

pital ship at Casablanca and Japan, and our instances of bacillary dysentery were tremendous. It mostly affected men who were active in the harbor. In fact, one ship was so badly disabled I don't think it could have pulled up its anchor, and we found—it was our practice at least to use sulfadiazine, which gave excellent results, and if that didn't work we had adequate laboratory facilities, and everything that a hospital should have. If the stools did not become negative through sulfadiazine we then used sulfanilamide, and between the two we were able to clear up every case practically and there was no mortality, although some of these fellows were passing pure blood for several days. But I would like to ask Dr. Keefer this question: We treated a great many cases of syphilis with penicillin and, of course, in those days the dose, as I remember it, was 1,400,000 units given over a period of seven and one-half days at three hour intervals. I would like to ask if in his opinion according to the present knowledge, that dose was adequate, so that these cases won't have more trouble in the future?

I would also like to ask Dr. Keefer, what is the outlook on the commercial supply of streptomycin, whether we are going to get a more adequate supply?

PRESIDENT STREIGHT — Any other question?

DR. RICHARD S. GUBNER — Increasing resistance to streptomycin occurs in many organisms in vitro. Does this occur in human tuberculosis, and if so, in view of the protracted treatment in tuberculosis, is it likely that this constitutes a practical limitation to the usefulness of streptomycin in tuberculosis? Have any studies been done which would show that that organism is recovered from sputum after streptomycin has been carried on for a month or two by increasing resistance over the initial resistance? Dr. Keefer indicated that some evidence had been presented that sulfa drugs were of some value. I would like to ask Dr. Keefer what is the present status of the sulfa drugs, such as, sulfadiazine?

DR. RALPH R. SIMMONS — I would like to ask Dr. Keefer if he will discuss with us by what tests we can determine when an individual has had sufficient treatment in cases of syphilis to render him safe as an insurance risk. I appreciate the fact that I am asking for that probably ten years before I should, but we are going to have to pass on these people in the intervening ten years.

One other thing that I would like to ask for help on is that not too long ago one of our officers developing a sore throat, bought some penicillin in tablets or lozenges. The package said definitely they should be dissolved on the tongue, but being one of those enthusiastic individuals he chewed it vigorously until he had consumed some three or four, to find out a few hours later that he had difficulty in his speech, which he attributed to the tablets. This difficulty persisted for about twenty-four or thirty-six hours and then it seemed to disappear. I would like to have some help on that problem.

PRESIDENT STREIGHT — Are there any other questions?

DR. JOSEPH W. JOHNSON — I would like to make an observation as to diphtheria and ask Dr. Keefer a question in regard to the problem of diphtheria that we encountered on the continent. We saw many men there who had acute diphtheria. They were given sulfonamides and were occasionally given penicillin. Many of those men came down with the most serious cases. Now it seems to me, as he implied this morning, we have had that type of diphtheria turning up in this country, and if we injudiciously use sulfonamides and penicillin without adequate bacteriology, we are going to run into trouble. The reason I stress the diphtheria problem is because it is quite different. Many of the cases looked like streptococcus for the first twenty-four hours. In Florence, Italy, 90% to 95% of the complications were polyneuritic.

The last question I want to ask Dr. Keefer is: Are the sulfanilamides and arsenicals used in syphilis effective in the diphtheritic groups? We had great difficulty with them in

trying to clear up carriers. I am leery of both of those agents in handling the problem of diphtheria.

DR. KEEFER—With respect to the question of dosage schedules in the treatment of syphilis, at the present time, the Committee on the Study of Syphilis of the United States Public Health Service, is continuing the work that began under the Committee on Medical Research of the OSRD. Perhaps many of you know that they set up a program which is nationwide on the basis of different dosage schedules. Originally Dr. Mahoney suggested the dosage schedule of 1,200,000 units given over a period of seven and one-half days in sixty equally divided doses. Other dosage schedules have been included of 2.4 and 4.8 million units given in a divided dosage over seven and one-half days, and the time dosage relationship has been changed from every four hours to every three hours to every two hours. While it is too early to make any statements about what is the best time dosage relationship and what total amount would give the best results, it is the opinion of Dr. Mahoney and his group that they have obtained the best results on a two hourly schedule using between 3.6 and 4.8 million units in the seven and one-half day period. All of these patients, of course, are followed carefully by periodic serologic examinations, and this is continued for a period of two years after treatment. In the cases of serologic relapse they have assumed in many instances they were relapses instead of reinfection. In some instances they obviously were reinfection.

I do not believe it is possible now to make any specific statements concerning the time at which these patients should be followed before one could be certain that they are safe risks. Certainly if the serologic reaction becomes negative and does not relapse within a year, one can be assured that such a patient would seem to be free of infection.

With respect to the outlook of streptomycin production, I don't know very much about the details, because I only know what I am told, and the Civilian Production Administration,

on the basis of information available to them, is of the opinion that there will be an increase in the present production about the first of the year, barring strikes and other things that have definitely delayed the production program.

It has been determined that tubercle bacilli are becoming increasingly resistant to streptomycin. Dr. Yeomans, of the Northwestern University, made a study of this in the cases that have been followed at the Mayo Clinic. So we do not know the meaning of this increase in in vitro resistance of tubercle bacilli following treatment. We simply have to follow these cases with great care and attempt to determine what this increase in in vitro resistance actually means.

I cannot answer the question of the difficulty in speech following the use of penicillin lozenges. Penicillin lozenges are of various types, of course. There is one that is incorporated into chewing gum or into paraffin, in which penicillin is liberated from the paraffin as it is chewed. The other, of course, is bound with a buffer, usually sodium citrate, in the form of tablets, which are to be swallowed. If penicillin is used in the form of tablets there is only one thing to remember and that is that at least five times as much should be used at a single dose or five times as much total daily dose as is usually required when given parenterally.

With respect to diphtheria, of course, the treatment of diphtheria continues to be anti-toxin. Penicillin has no effect on the toxin of diphtheria or the lesions that develop in man as a result of intoxication.

With respect to carriers, it has been found in our clinic in Boston that if adequate amounts are administered that is, at least 100,000 to 150,000 units a day for ten to fourteen days, the carrier state can be greatly reduced when you start treatment during the active phase of diphtheria. If acute diphtheria is treated with anti-toxin and penicillin the number of carriers at the end of fourteen days is greatly reduced. When carriers are treated, on the other hand, then the problem is

very much more complex. Here even when large amounts are given for a period of fourteen to twenty-one days, the carrier state may not be eliminated. Why that should be so we do not know, but if you are going to use penicillin in the treatment of diphtheria in the acute phase it should be used with anti-toxin. It should be continued from ten to fourteen days, and with the carrier certainly that is the minimum period of treatment — not just a few days.

DR. ARTHUR J. MCGANITY — Mr. Chairman, if I may ask just one question. In our last meeting we heard a great deal about sulfonamide sensitivity and we heard warnings about it. Since that time there has been an enormous amount of experience in the armies all over the world in the use of these drugs for very many conditions. Has Dr. Keefer anything to add to our knowledge about that now? Are we perfectly safe in taking people who have been treated with sulfonamides for a short period after they have recovered from the disease for which they were treated?

DR. KEEFER — I believe that question was raised the first time I had the privilege of talking to this group in Boston. One hears a great deal about patients who cannot take the sulfonamides, and I think that it is true for the reason that a certain number of patients will develop skin eruptions and fever if they are sensitive to the sulfonamides. I do not know that it has produced any permanent harm. Certainly, if cases of permanent tissue damage are being produced by the use of the sulfonamides they must be extremely rare, for the reason that literally thousands of tons of sulfonamides have been taken by the people of this country in the last few years. In patients who develop renal complications from the sulfonamides, in our own experience, it is usually all or none. They recover completely or they may actually die, but one should not be afraid to use the sulfonamides because of renal complications because they can be prevented if you see that the patient gets enough fluid. If he puts out 1,500 cc's of urine a day you don't have to worry about renal complications. The difficulty is

that some patients don't like to take fluid if they are taking sulfonamides for a sore throat, or if they are dehydrated and have been vomiting before the sulfanilamide is started then they may get into trouble, for the reason that the sulfonamide is being given to a patient who hasn't established a free flow of urine. We haven't had one instance of renal complication due to sulfonamides in our clinic in five years, and the reason is that the drug is never started until the patient has a free flow of urine. So that renal complications in our experience are rare.

PRESIDENT STREIGHT — The next order of business is to ask the experts to question the experts. Dr. Albert J. Robinson, Medical Director of the Connecticut General Life Insurance Company of Hartford, Connecticut, will take charge of this part of the program.

Dr. Albert J. Robinson assumed the Chair . . .

OPEN FORUM
on
PROBLEMS OF MEDICAL SELECTION

ALBERT J. ROBINSON, M. D., *Moderator*

Connecticut General Life Insurance Company

When your Program Committee asked me to be Moderator for this Open Forum, I accepted the assignment because I believe every member of our Association should be willing to do such extra-curricular work as he may be asked to do by our Program Committee, and second, because I am personally enthusiastic about the idea of an Open Forum on our program.

Notwithstanding the shortcomings of your Moderator, which will be all too apparent as we progress, I believe we can all derive much benefit from an Open Forum. We come to these meetings to find out what others are thinking and how they are meeting their underwriting problems. A Forum such as this affords every member of the Association an opportunity to get a cross-section of opinion from a group of Medical Directors and their associates, who are collectively the best authorities in this country on medical history and impairments as they relate to insurability.

I wish to express my appreciation to all those who wrote to me suggesting topics. There was a wealth of excellent material. My greatest task was to make a selection and limit the number of topics to fit our time schedule.

I have chosen six topics, some of which are currently controversial. They all present underwriting problems to Medical Directors and their associates. They are:

1. "Insurability of the Diabetic."
2. "Under what circumstances do you insist upon an attending physician's statement? How do you obtain it and what do you pay for it?"

3. "The Prudential concept of the necessary ratings for personal or family history of pulmonary tuberculosis or exposure to this disease."
4. "What constitutes more than one attack of duodenal ulcer?"
5. "Should a QRS of less than 5 mms. in an EKG which is otherwise satisfactory ever be accepted standard, or if offered sub-standard, at what rating?"
6. "Diverticulosis and Diverticulitis."

Because we are going to be pressed for time to cover this number of suggested topics, it is my desire to have you confine your discussion to facts, to give in a concise manner your specific opinion and current company action. I therefore ask that you confine your remarks to the question under discussion. I am sure that everybody will appreciate brevity and strict adherence to the point under discussion. If it becomes necessary to close the discussion on any one of these topics before we have exhausted all of the good ideas which might be presented, I know you will realize that I do so in order that we may cover the six topics I have mentioned. If we have any time left after we get over these topics, we will pick up your unanswered questions at that time.

Without further ado we shall go into the question of the insurability of diabetics. Inasmuch as Dr. W. J. Allison, who is medical Director of the Southwestern Life of Dallas, Texas, has, I believe, accepted diabetics over a longer period than any other company that I know of, I thought that he would be an appropriate individual to open the discussion on this subject. Dr. Allison!

DR. WILFRED J. ALLISON—The Southwestern Life has been accepting the careful, controlled diabetic since December, 1940. Originally we did not take any diabetics below age 17 and accepted from \$1,000 to \$20,000 at Table D on Twenty Payment Life or higher premium plan on individuals whose contract would endow or mature not later than age 55. At this time we did not require either electrocardiogram or X-ray of the chest. In recent years, however, we have been taking diabetics for a minimum of \$5,000 and a maximum of \$30,000 from ages 10 to 60 on Ordinary Life or higher premium plan

at a rate not less than 200 per cent of the normal rate. Whether or not on insulin, these individuals must have periodic check-ups by a physician well qualified to treat diabetes, he or she must be within the weight limits for standard insurance, must not require over 60 units of combined insulin per 24 hours, must otherwise be in good physical condition and must present a normal current electrocardiogram and chest X-ray. We prefer to postpone the newly discovered diabetic for a minimum of six months to determine the severity of his disorder, and whether or not he proves intelligent and co-operative.

Diabetics now come to us by trial form for a minimum of \$5,000 insurance with a resume from the attending physician. If recent fasting and postprandial variations have not been determined, we may require one or more blood samples but do not require glucose tolerance test. For example, if a co-operative diabetic has maintained satisfactory regulation for the past several years on 40 units of protamin zinc and 10 units of regular insulin daily and both he and his doctor know that either he is continuing with a sugar-free urine, or spills only small amounts occasionally, we should ask for no more than a fasting and a two-hour postprandial determination. If the attending physician has made such studies within the past year and they are satisfactory, we should not even require these. No doubt we have irked some busy physicians in asking for these studies but I think we have also made them pay closer attention to some of their diabetic patients, especially to those who have glycosuria when examined for life insurance. If a physician has never determined the renal threshold for his diabetic patient, we suggest that he do so. For this reason alone we ask laboratories, in doing glucose tolerance tests on questionable cases, to check blood values with time intervals and urinary excretion so that the attending physician will know approximately the level at which the patient begins to spill.

We inquire of all attending physicians as to whether or not they have made a cardiogram or chest X-ray recently and if

such studies have been made, we ask to have them sent to the Home Office. I recall two instances in the past eight months where statements from attending physicians outlined the patient's satisfactory progress regarding his diabetes and further communication before authorizing examination revealed quite abnormal electrocardiograms already in the patient's file.

We do not issue accidental death or disability benefits to diabetics. If any other ratable impairment makes the total over 300 per cent, we make no attempt to classify further. We do not accept an individual who has had over one episode of threatened coma since beginning treatment.

We have also insured a few individuals classified as potential diabetics. Such applicants were found to have transient glycosuria, a high postabsorptive curve and peak values at either one-half or one hour of 200 mgs. per cent and higher, but also showing rapidly declining curves with two hour values at 120 or below. Realizing the limitations of the oral glucose tolerance test, we are aware that some non-diabetic individuals may show a declining curve but still have a two-hour value of more than 120 mgs. per cent. We recognize that a very mild diabetic may exhibit extreme values in performance of this test. In questionable cases we may extend the test to three or even six hours. For purposes of selection we have not discarded the idea that the peak of the curve is of no significance. In some instances we have either the attending physician, the examiner or the laboratory instruct the applicant to avoid fats and to take 200 gms. of glucose in excess of regular daily diet for two days prior to repetition of the test. No doubt we have made some errors in classification and will continue to do so. These individuals whom we have accepted at Table B have at least a temporarily impaired glycogenesis and therefore an impaired glucose metabolism. We have not studied liver function in this connection and have not used the intravenous glucose tolerance test.

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We have accepted a few individuals who were said to have hyperinsulinism—all at the standard rate. So far we have encountered none with fasting or six hour values below 70 mgs. per cent.

In handling to date slightly less than 200 diabetics, we have accepted approximately 75 per cent of those recommended by their physicians. The average age at entry is 38. One at age 10 and one at age 17 are the youngest. The average age at entry of a very few potential diabetics is 41. So far there has not been a claim in either group.

I have no doubt that in due time more significant figures from larger companies will be available and that a more scientific handling of the problem will be in general use.

CHAIRMAN ROBINSON — Dr. R. C. Montgomery, Medical Director of the Manufacturers' Life of Toronto will continue the discussion. Dr. Montgomery!

DR. RICHARD C. MONTGOMERY—Dr. Robinson said something about two or three minutes, so I therefore took the liberty of setting down what information we had on paper, and this has been given to you.

I would like to congratulate Dr. Allison on his summary of his handling of these people. We follow pretty well the same practice that he does. When we decided to go into this field we did not have anything to go on and we merely picked out the figure of 200% such as he has used. The actuary thought that \$10 was a good round figure and, therefore, we said that anybody we take we will charge an extra premium of \$10 per \$1,000 and this we have followed, no matter what the plan. Now I have set down on paper, as I have said, what we have done so far and you can read most of that stuff at your leisure.

Unfortunately, there is nothing there to help us very much as no one has died. However, that doesn't prove very much. There is one interesting factor which I haven't recorded, and that is concerning blood pressure. We decided that anyone

MANUFACTURERS LIFE INSURANCE COMPANY UNDERWRITING DIABETICS

HISTORY

The underwriting of diabetes was commenced in 1939 but the majority of this business was written during the last five years.

UNDERWRITING BASIS

Age 25-60 male and female who have been under regular supervision for at least three years and are well stabilized.

Plan — Ordinary Life par or better.

Extra — minimum \$10.00 no matter what plan

Age 25-30 — extra \$15.00 per M

" 30-45 — " \$10.00 " "

" 45-60 — increased over the \$10.00 by \$1.00 for each year of age over 45.

Amount — \$25,000.

Special requirements — Ekgm. and 6' plate if 50 years of age or over, or if amount is over \$10,000 at any age.

PROCEDURE

Branch Office submits applicant's questionnaire as well as the inspection tickets.

Company has a questionnaire completed by the applicant's doctor and orders special diabetic inspection.

Company authorizes examination, quoting probable extra.

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THE MANUFACTURERS LIFE INSURANCE COMPANY
Statistics on Business in Force on Diabetics as at September, 1946

Number insured — 280 Amount — \$2,750,900		Total extra premium — \$63,150 Average — \$12.00 per M	
AGE		OCCUPATION	
20-29	29 policies	Executives	— 116
30-39	117 “	Physicians	— 24
40-49	102 “	Salesmen and agents	— 22
50-59	28 “	Retail stores	— 20
60-	4 “	Accountants	— 14
Insurance in force largest in 40-49 group — \$1,130,000.		Lawyers	— 12
		Chemists	— 7
		Teachers	— 5
		Engineers	— 6
		Watchmakers	— 4
		Others	— 50
NET WORTH OF APPLICANT		INCOME OF APPLICANT	
under \$10,000	— 91	under \$10,000	— 177
over \$10 - 50,000	— 138	over \$10,000	— 103
over \$50,000	— 51		
UNITS OF INSULIN TAKEN			
Units	Number of Applicants	Average Units of Insulin	
0-25	128	6.8	
25-50	96	37.6	
50-	56	62.5	
Included in first group are 76 who took no insulin. Largest amount taken — 88 units. Overall average — 28.5 units.			
WEIGHT		DURATION OF TREATMENT BEFORE INSURANCE	
Overweights	— 104	shortest	— less than 1 year
Underweights	— 171	longest	— 30 years
Mean weight	— 5	average	— 9 years
This includes three—50 lbs underweight one—60 lbs. overweight			
DECLINED CASES			
Reason	Number	Reason	Number
Not well supervised	11	Age	1
Blood pressure	6	Heart murmur	1
Ekg.	6	Coronary	1
Habits	3	Enlarged heart (x-ray)	1
Insulin — too much	3	T. B. chest	1
Albumen	2	Others	4

THE
MANUFACTURERS LIFE
INSURANCE COMPANY
Head Office - Toronto, Canada

DIABETIC QUESTIONNAIRE

To be Completed by Applicant

NAME..... Date of birth.....

RESIDENCE.....

1. Occupation..... Former occupation.....

2. Height..... Weight..... Weight two years ago.....

3. Date Diabetes diagnosed.....

4. Name and address of Doctor making the diagnosis.....

5. Are you receiving treatment or under medical supervision now?.....

6. Give name and address of Doctor.....

7. Do you ever stop the insulin or go off diet.....

8. Is urine sugar free? (a) Now..... (b) Always..... (c) Date of last test.....

9. Have you had any blood sugar estimations done?.....

10. If so, when and what were the fasting estimations?.....

11. What is the diet at present? Protein.....gms. Fat.....gms. Carbohydrates.....gms.

12. Is the diet weighed or estimated? (a) weighed..... (b) estimated.....

13. State amount of insulin taken daily.....units. Type—Plain.....units.
Time of administration..... Protamine Zinc.....units.
Globin.....units.

14. Have you ever had any infections, such as boils, abscessed teeth, tonsillitis, etc.?.....
Specify.....

15. Have you ever had any eye trouble?.....

16. Have you ever had heart trouble?.....

17. Have you ever had high blood pressure?.....

18. Have you ever had any recurring or prolonged illness?.....

19. Has an electrocardiogram been taken?.....Date.....By whom.....
(If electrogram has been taken in the past, submit copy which will be returned.)

20. Was the E.K.G. normal?.....

21. Has an x-ray of the chest been taken?.....Date.....By whom.....

22. Was the x-ray normal?.....

23. Amount of insurance contemplated?.....

Date..... Signature applicant.....

Dr.

I hereby authorize THE MANUFACTURERS LIFE INSURANCE COMPANY to obtain from you an abstract of your records, including treatment, diagnosis and prognosis.

Thank you, for this courtesy.

Date.....

Witness.....

THE
MANUFACTURERS LIFE
INSURANCE COMPANY
Head Office: Toronto, Canada

DIABETIC QUESTIONNAIRE

To be Completed by Attending Doctor

NAME Apparent age

RESIDENCE

1. Occupation

2. Height Weight Weight two years ago Weight 5 years ago

3. Does this patient visit you regularly for supervision? Date of first consultation

4. What is the diet at present? Protein gms. Fat gms. Carbohydrates gms.

5. How is the diet measured? Weighed Estimated units.

6. How much insulin is taken daily? units. Type—Plain units.

Time of administration Protamine Zinc units.

7. How much insulin was taken previously? a year ago 2 years ago units.

8. Has the patient had any insulin reactions? When?

9. Does the patient follow your advice consistently?

10. Over a period of years has there been a gain or loss in tolerance?

11. Is the fasting urine free of sugar?

12. Fasting blood sugars. Give dates and estimations of recent tests

13. Are there any changes in the eye grounds?

14. Is there any evidence of (now or in past):

(a) Pulmonary tuberculosis?

(b) Heart disease?

(c) Any recurring or prolonged illness?

(d) Infections such as boils, infected teeth, tonsils, etc.?

(e) Any abnormality of palpable arteries?

15. Is there a good pulsation in posterior tibial and dorsalis pedis arteries?

16. What is the highest blood pressure reading recorded? When?

17. Has an Electrocardiogram been taken? Date By whom

If cardiogram taken, submit copy. (This will be returned.)

18. Was the E.K.G. normal?

19. Has an x-ray of the chest been taken? Date By whom

20. Was the x-ray normal?

21. Do you consider the patient a mild, moderate or severe diabetic?

22. Further comments

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Date

Form 1180C 1a 12-65 T. 49209

Signature attending physician

Address

qualifying as a diabetic should have no impairment other than, perhaps, some over-weight, and we, therefore, set the limit of blood pressure as 140/90. In looking over our blood pressures I find that we did take one individual whose blood pressure was 145/85. The lowest recorded was 90/60; the average 121/76.

I have included in these papers the questionnaires which we used. We first get information from the applicant himself because if he is a well regulated diabetic he has a perfectly good idea of what he is doing, and we sometimes were able to weed out individuals we felt were not insurable. When the replies to the applicant's questionnaire are satisfactory we send our questionnaire to the applicant's doctor and when that is received we are in a position to advise our branch office as to the amount of extra which will be charged. Whether \$10 is right or not, I don't know—that remains to be seen, I guess. However, we propose to carry on in that way. These diabetics are taken pretty well on an individual basis. We look at each one carefully and if there is any factor whatever which we think is important, we change the plan from Ordinary Life to Twenty Years or increase the extra from \$10 to a higher rate.

I went over the declined cases and found that we had declined about 20% of those submitted to us. Of these declined cases, you will note, we declined 11 for not being well supervised and about one-third of the total we declined for blood pressure and other cardiac abnormalities.

Another thing that I think is important is that we ask for a cardiogram six foot plate on everybody who wants more than \$10,000 or who is 50 years of age or over. Thank you.

CHAIRMAN ROBINSON—I believe that recently the Lincoln National decided they would experiment in the field of insurance in diabetics and I, therefore, asked Dr. W. E. Thornton, Medical Director of the Lincoln National Life if he would make one or two comments to us regarding their attitude and current practice. Dr. Thornton!

DR. WALTER E. THORNTON—It is but a few weeks ago that the company which I represent issued its first policy to a known, frank diabetic. This period is so short that I should stand in some embarrassment, and I hope you will believe me sincere when I tell you I would not be doing so had not the Chairman of this forum, Dr. Robinson, invited me to make these comments. However, we have given considerable thought to the impairment over a period of months and have formed some opinions which I trust may be of interest to you.

I think when a company embarks upon the field of insuring frank diabetics, they should agree that, in this insulin age, well treated diabetes has an excellent clinical prognosis. The once familiar diabetic coma is now rare. There is still some extra mortality from degenerative disease but even this is believed confined to those cases, the treatment of which is indifferent and careless.

From such a theoretical background the group indices for purposes of insurance classification seem clear.

- (a) Definite clinical or laboratory diagnosis, otherwise the case is treated as one of glycosuria.
- (b) Classification as to severity is judged
 - (1) from the daily insulin requirement (if any)
 - (2) from the diet formula
- (c) The expertness of the physician attending as judged from his qualifications.
- (d) The care and constancy in the treatment as reported by the attending physician.
- (e) Such evidences of complicating degenerative disease as may be found or suspected from findings in the examination or other parts of the application.

If we, as an individual company, are making any special contribution to the handling of diabetics as insurance risks, it is in our efforts to simplify the procedures involved. We are not using special blanks for the purpose. We are attempting to handle these cases as we handle most impairments as

far as forms are concerned. The critical information I have referred to above, we get by adding only four special questions to our regular Family Physician blank. Thus:

- (1) The total amount of insulin (all types) taken per day?
- (2) Diet formula?
- (3) Is the patient co-operative and meticulous in treatment?
- (4) How long has he been under the care of a physician for Diabetes Mellitus?

Already we have found that we must make a change. We must reword question No. 2. Too often our informants are giving only the caloric contents of the diet. We must ask them specifically for the protein, fat and carbohydrate distribution.

That there will be further changes, I have no doubt. Our efforts to be brief may defeat our purposes. We shall know in another few months.

In conclusion I would like to join in complimenting Dr. Montgomery and Dr. Allison in their courageous pioneering in a difficult and complex impairment.

CHAIRMAN ROBINSON—I believe it was in June of this year that I announced to the Connecticut General Field Force that we would consider diabetics and I told my very able associate that it was his problem to tell us what he is doing with them. Dr. Barker!

DR. NORMAN J. BARKER—During the past year, the Connecticut General announced a program of substandard life insurance for diabetics, male and female, ages 10 to 60, on permanent plans of insurance without benefits and for amounts of \$2,500 to \$25,000.

We require a preliminary inquiry form and a diabetic questionnaire completed by the applicant which includes an authorization to the attending physician. If we are interested in

giving the case consideration, we communicate directly with the attending physician and ask him to complete a questionnaire for which we allow a fee of \$5.00. If on this evidence we are willing to entertain an application, we authorize an examination and make an offer covering the amount, plan of insurance and approximate rating.

For underwriting, we divide diabetics into four groups.

Group I—Good economic grade, family and personal history and habits; average weight, blood pressure under 140/90; diabetes of comparatively short duration and mild in terms of daily insulin requirements; faithful and intelligent use of insulin and diet which must include an adequate amount of carbohydrate. The urine must be sugar-free most of the time with a satisfactory fasting blood sugar level; no history of coma or other diabetic complication and good peripheral circulation. Applicants under age 35 must have had an X-ray of the chest and over 45 an electrocardiogram within a year. All must be under regular medical supervision. Applicants who qualify in this group are offered any of the Company's permanent plans of insurance with a mortality rating of 200% to 250%.

Group II—Diabetics who fail to measure up to the standards established for Group I in possibly one or two respects are offered any of the Company's permanent plans of insurance at mortality ratings of 300% to 500%.

Group III—Applicants with long standing diabetes but who are otherwise acceptable may be offered a plan of insurance maturing in fifteen years at ratings of 300% to 500% and a limit of \$10,000.

Group IV—We do not feel that we can insure diabetics of poor economic or industrial grade, with poor family or personal history, diabetic complications or other rateable impairments; or those with very severe diabetes and little or no medical supervision; who are careless in the use of diet or insulin, with high fasting blood sugars or constant glycosuria in large amounts indicative of poor control.

In such a new field, we have obviously been obliged to set up somewhat general rules for the underwriting of diabetics. With experience, we hope to be able to establish more definite standards of selection. In the very short time our program has been in operation, it has been possible for us to offer life insurance to a large number of diabetics and on a basis which we feel is fair both to the policyholder and to the Company.

CHAIRMAN ROBINSON—My watch says that we have spent twenty-three minutes on it and the President reminds me that we have spent twenty-three minutes. I would like very much to have an open discussion of this question of diabetics. I hope we will have it at the end of our forum but in order to cover the topics that we propose to cover I am going to take the liberty of going on to the next question "*Under what circumstances do you insist upon an attending physician's statement?*" "*How do you obtain it and what do you pay for it?*" and I have asked Dr. Chester T. Brown, Medical Director of the Prudential, to give us their practice. Dr. Brown!

DR. CHESTER T. BROWN—The practice to obtain an attending physician's statement is one of a number of procedures to procure an accurate knowledge of an applicant's insurability. No fair-minded person should object to the procurement of complete and dependable information which has for its objective a protection of the company on the one hand, and a fair and willing attitude on our part to clear up doubtful features which otherwise might result in unfairness to both the applicant and the agency.

For approximately fifteen years, we have been asking for attending physician and hospital statements, paying respectively \$2 and \$5 for them, and the forms we use indicate the fees which will be paid. With the thought that one cannot predict from the answers to questions on the application whether the answer refers to a trivial or important cause for medical attention, a rule was set requiring physician's statement for

amounts of insurance of \$25,000 or more for applicants of 40 and under, and for amounts over \$5,000 for applicants over 40 years of age. As the years passed, our experience encouraged us to believe that it would be advantageous to broaden these limits of requirements, so that now statements are requested irrespective of amounts and whenever so decided.

There are some sorts of answers in the application which seem, automatically, to cause physicians' statements to be requested. Two such statements by applicants are outstanding in frequency and often presage an unfavorable circumstance. These are "just a physical check-up" and "for a cold," when the actual symptoms or findings turn out to be chest pains, and moderate hypertension or similar ominous circumstances.

Statements which are of most value are those which I call "spontaneous," that is, those which we request directly from the Home Office, the request being addressed to the attending physician and which come directly to us without the agent's or applicant's assistance. Otherwise physicians' statements are apt to be less accurate and modified on the side of gentleness. Statements in the form of an attending physician's letter written prior to and accompanying the application to the Home Office are likely to be of an over-benign quality.

Naturally, the request for statements is likely to delay action on an application and much time could be saved were the request to go directly to the agency. We avoid such a procedure as much as possible, resorting to agency assistance only after a second request to the physician fails to bring in a statement. In the large cities where we have Medical Referees, statements usually are requested by the Referee who has an opportunity to telephone the physician and get the substance of the applicant's history.

Unquestionably, these two to three hundred statements which we receive monthly are a benefit to our mortality experience, giving us knowledge of impaired risks and strengthening our assurance concerning good ones.

CHAIRMAN ROBINSON—There is a rather small company down here in New York. They call it the Metropolitan. I asked Dr. Bonnett, their Medical Director, to give us his practice with respect to Attending Physicians' Statements. Dr. Bonnett!

DR. EARL C. BONNETT—In the Metropolitan Life Insurance Company, we insist on such statements:

1. To establish definite dates, durations, and diagnoses when information secured from the applicant is contradictory or not clear-cut.
2. When a person has had a "check-up examination" and it does not appear from the application that this is a routine procedure.
3. When a person has had an operation, the cause for which is:
 - a. "Unknown"
 - b. "Minor" but the resulting incapacity is disproportionate to the alleged nature of the operation.
 - c. Apparently of such a nature that the surgeon prefers not to disclose it to the applicant. These include stomach, bladder, rectal, or serious gynecological operations particularly at the older ages. In such cases, we prefer hospital statements with the additional information about the laboratory and pathological findings which they should contain,

Method: We send our form to the District Office with the request that the Agent secure the applicant's authorizing signature and then mail the form to the doctor or hospital making sure that the address of the doctor or hospital as taken from the application is accurate. The form, when completed by the doctor or hospital authority, is returned directly to the Home Office in a self-addressed stamped envelope. Our fee for this service is \$2, but where we know that hospitals have a different standard charge, that fee is paid.

In our experience, the proffered fee seems acceptable in the majority of cases. If we were to consider any increase in the

fee, we believe that the fee paid for a hospital statement should be \$5 and for a physician's statement not over \$3.

We occasionally write directly to an applicant asking him to provide us with a statement from his doctor, or a diagnostic film, and in such cases, we usually stipulate that the information is to be received at no expense to this Company.

We receive about 75% of the statements requested, but the delay is considerable. To be worthwhile and justify the time and expense of securing it, supplementary information must be detailed, accurate, and promptly forthcoming. One category of such supplementary information is a statement from a doctor's or hospital's records.

CHAIRMAN ROBINSON—It seemed to me that we might go to the Mid-West and pick on somebody that wasn't quite as large as the Metropolitan, so I ask Dr. Simmons, who is Medical Director of the Equitable of Iowa, to tell us his practice. Dr. Simmons!

DR. RALPH R. SIMMONS—I surmise that I was asked to discuss this subject since our Company uses very few forms for obtaining information from the applicant's personal physician.

We have felt that our best approach to the personal physician was by a "tailor-made" letter signed by a Medical Director. We feel that any request to the personal physician should be predicated on a real need for additional information and that this request should never become a routine procedure to be put into the hands of a clerk. We do not encourage our General Agent or Soliciting Agent to request information from a personal physician, in anticipation of our needs, since we have found that these requests rarely cover, in sufficient detail, our underwriting needs.

We ask for reports where additional information is required regarding the diagnosis of a condition, the dates upon which this condition occurred, where additional clarity is required for the proper classification of the disorder, where we feel that an additional check is required because of a possible

inaccuracy in the applicant's declaration, to obtain details regarding an impairment suggested through outside sources and not reported by the applicant or finally, to obtain reports on special studies or consultations such as electrocardiography, X-ray, etc.

I am especially interested in the activities of the committee headed by Dr. Ungerleider, in suggesting standardized forms of inquiry as requested by the American Medical Association. I believe that this committee has a difficult task to perform, since as stated earlier in my remarks, we have not, as a Company, subscribed to the use of forms in obtaining information from the applicant's personal physician. We have not been able to employ any general form for letters of inquiry that we personally felt would be effective in all cases. We trust that the Committee will be successful in its efforts and we will be glad to use such standardized forms as they may recommend.

CHAIRMAN ROBINSON—The boys are really doing pretty well. I told them, "Don't you dare to come up here and talk longer than five minutes!" I am going on, with your permission to the next topic which I have outlined, namely, the "Prudential Concept* of the Necessary Ratings for Personal or Family History of Pulmonary Tuberculosis or Exposure to this Disease" and I have asked Dr. H. W. Dingman, Medical Director of the Continental Insurance Company, to give us his comments. Dr. Dingman!

DR. HARRY DINGMAN—The Prudential has done the insurance fraternity a service in challenging our concepts of how to appraise the tuberculous. The Prudential knows, and you know, and I know that tuberculosis was the No. 1 cause of death in the 20's, and the No. 2 cause in the 30's and the No. 3 cause of death in the 40's. We can well give a little time and thought to this subject. Their rationality is in brief that

*The Record, American Institute of Actuaries, Volume XXXIII, Pages 319-93; XXXIV, Pages 92-113

tuberculosis, being an infection, is a contagion, and the whole hazard is in *contact*. Forget age, forget family history, forget recency of attack. It's infection.

The second point is that the diagnosis can be made by X-ray and there, their case rests. They want us to pin our faith on X-ray.

It seems to me very dangerous to forget age. Age proves, if nothing else, that survival to middle age means ability to defeat infections that kill the weak. My grandfather, if living at his age, renounces no rights to acquire tuberculosis. But he resists the disease better, and longer, and tuberculosis is slower to kill him. To say, "Forget Age" sounds illogical.

Weight proves, if nothing else, that overweights, on average, are better nourished than underweights. Undernourished persons pick up tuberculosis or any other infection, more readily than well nourished persons. Sound logic tells you the which. And experience. Experience is abundant, insurance experience especially.

Forget family history? At your peril. Few Man-O-Wars come out of truck horses. Few strong lunged persons out of weak lunged persons. And if you emphasize the environmental factor as compared to the hereditary, what then? Tuberculosis is a disease of the poor, poor in pocketbook, poor in physique. And those who are poor in pocketbook and physique come, on average, from parents who are poor in pocketbook and physique. If you argue contrarily, read insurance experience.

With regard to recency of history, by orthodoxy of experience, wait two years and make your appraisal. By Prudential heterodoxy, wait two years and reject, wait five years and reject, wait eight years and say, maybe, if the applicant had a moderately advanced case of tuberculosis. Continental solicits those rejects.

Prudential emphasizes that X-ray is an excellent adjunct to diagnosis. It is, but uncertain. "Suspicious film findings

must be corroborated by a positive tuberculin test and by positive bacillary findings." So said Hilleboe. "We have seen so many errors made that we now accept nothing except a complete examination of which the X-ray film is only a part." So said Myers. Many lung lesions cause no shadows because they do not calcify. Calcified lesions, easily seen, are relatively unimportant. Soft lesions, easily missed, are truly important. Histoplasmosis, a pulmonary fungus infection, causes lesions often mistaken as tuberculous. Myers sees the hazard of misinterpretation. Perhaps we should also. It was Myers, also who said that chest examinations of children are almost fruitless.

X-ray is an excellent adjunct to diagnosis. But expensive. Multiple pictures are oftentimes necessary. Prudential can afford it, to the profit of all of us. Of 16,150 National Guardsmen and Selective Service registrants, average age 25, in southern New York, 70 had possible active tuberculosis—230 chest studies to uncover one case, many requiring repeat pictures.

Prudential concept of insurability of the tuberculous has jolted our complacency. Annie May Lyle has presented the case magnificently, as of course she would. Even though wrong, if wrong they are, Prudential has made us think. Many of us do too little of that sort of thing, seems like.

CHAIRMAN ROBINSON—I have asked Dr. Weaver, Associate Medical Director of the Penn Mutual, to comment further on this topic. Dr. Weaver!

DR. ROBERT L. WEAVER—I quite agree with Dingman in many respects. I think he has covered the subject very well. I think that Miss Lyle has done more than just make us think. I think Dr. Homan in 1936 warned us of the changing concepts in tuberculosis. I think Miss Lyle blasted us again in 1944. I think it is time that we changed our ratings.

Unfortunately, I don't feel too well qualified to comment on the Prudential ratings, as I have not been in a position to

apply them and I have not made any comparative studies. I can only tell you what we are doing at Penn Mutual, which is probably a great deal more conservative than many of you are doing today.

First of all, in every case as far as possible we are getting X-ray films, probably a series of X-ray films, and we draw no line as to the amount. We may get them on a \$1,000 case as readily as on a case involving \$25,000 or over. We believe it pays. Naturally, we are not accepting any cases where there is a multiple involvement, that is, of different organs. In the multiple cases and the moderately advanced cases we are allowing credits for X-ray films which show calcification over and above the old ratings which were proposed according to age, build and history. I don't know that this is too good a plan but it is the plan we work on. Personally, I am greatly in favor of the Prudential ratings, and I shall be interested in seeing mortality studies in the future.

In regard to exposure, family history, we naturally are considering exposure, and only accepting children when exposure could not have taken place, where the disease was apparently arrested before the child was born, or it was stopped, or the child can show a negative tuberculin test or a negative X-ray. We are treating primary calcifications very similarly to the Prudential, debiting not at all for the isolated calcification. Where the calcifications are numerous and extensive the rating is about thirty-five. We are not accepting any lesions which in our estimation will rate over 125 additional mortality.

I think that pretty well covers our practice. I might add that we have been very fortunate in securing a series of X-ray films from attending physicians in hospitals; and on the subject of statements we do find that a personal letter to the doctor or hospital guaranteeing a fee is very productive. I am amazed at the response and the co-operation has been extremely good. Thank you!

CHAIRMAN ROBINSON—Continuing the discussion of the same subject, we will hear from Dr. Beard, who is Assistant Medical Director of the Mutual Benefit. Dr. Beard!

DR. J. RANDOLPH BEARD—I feel that the thoughts emanating from Dr. Kirkland's paper are pertinent and important. From a public health standpoint and the intricacies of statistical studies, it would appear that tuberculosis was a minimal hazard, for one is taking a cross section of population; a mortality rate. The figures presented by Lyle in 1944 on the Prudential study on cases of tuberculosis among their employees, and the Metropolitan studies along the same line, paint a gloomier picture. The fatality rate is high. I agree with Dr. Kirkland that insurance companies have placed too much stress on underweight at younger ages, and treated overweight, normal weight and the older age group too lightly in contact or exposed cases. The underweight individual is no more susceptible to tuberculosis than any other group, all other factors being equal. The Medical Impairment study of 1929 followed a traditional stage in the approach to tuberculosis. I definitely feel that another study is in order with an eye to the modern concept of the disease and the more up-to-date methods of treatment. The general population is better educated and the tendency is to make the diagnosis earlier. These facts should lead to some liberalization on the incipient and minimal cases. I do feel that X-rays are a necessity in evaluating applicants who have had tuberculosis or a history of exposure to the disease. In the latter group, I feel that more detailed information as to the extent, contact, surroundings and the like are also of primary importance. The tuberculin test is a medical nicety which we cannot expect in the insurance field until the populace has had a greater education in preventive medicine. Mass X-ray studies and tuberculin studies which are ever increasing will be of invaluable aid to underwriting, provided accurate reports are available. Since the Mutual Benefit Life Insurance Company writes only standard insurance, our present interest is twofold, is the applicant insurable

or not. We have adhered closely to a ten year waiting period, always call for a recent X-ray of the chest and obtain as many of the previous X-rays as possible. In applicants under 18, with a history of exposure, we decline until after the 18th birthday and also require an X-ray of the chest. A quick glance at the Prudential rating table gives the impression that it is rather severe. However, in light of the Prudential and Metropolitan studies on employees, the table is conservative. When one considers that these employees had the benefit of the best of care, the outlook for the run of the mill tuberculosis case is rather perilous.

In summary, I feel that mortality figures have possibly lulled us into a false sense of security. Dr. Kirkland's paper should certainly furnish food for thought for a more modern approach to tuberculosis underwriting, the necessity for a new medical impairment study, more attention to contact or exposure rather than build and age, and the absolute necessity for X-ray studies in the applicant with a personal or family history of tuberculosis. There are so many uncontrollable factors that each case has to be underwritten on its merits. I do feel that a revision of the overall picture in the light of new concepts might lead to some liberalization in certain classifications of the tuberculous applicant.

CHAIRMAN ROBINSON—I sense that there are very many questions in the audience but I am doing so well on my time schedule that I am going to ask you to give me the liberty of pushing this thing along. Our next question is: *"Should a Q R S of Less Than 5 mm. In An Electrocardiogram Which Is Otherwise Satisfactory Ever Be Accepted Standard, Or If Offered Substandard, At What Rating?"* It seemed to me that because Dr. Birchard, who is Chief Medical Officer of the Sun Life of Canada, is not only an experienced Medical Director but also an extremely competent cardiologist in Montreal, he would be a very good person to open the discussion on this subject. Dr. Birchard!

DR. CECIL C. BIRCHARD—There are two approaches in formulating an answer to the conundrum which may be accounted as plausible, one entirely empirical and based upon impressions gained from clinical observation, and the other deducible from accepted theories as to the how and why of the electrocardiogram.

First, one must ask the protection of a number of assumptions:—For instance, that the patient is of normal build and without deformity of the chest and that he is not unduly overweight; also, that the observer have available to him unipolar leads made from the chest wall, at least three of these, and made from positions 2, 4 and 6, or alternatively, from 2, 4 and 5. One does not demand unipolar leads made from the limbs though personally I would like to have them.

In any given electrocardiogram exhibiting low voltages of the Q.R.S. deflections of the standard leads, there are, I believe, certain attributes which modify the significance of the finding and these have to do with the normality of the Q.R.S. deflections and the T. deflections in the unipolar chest leads. Also, I should add, normality of the T. deflections in the standard leads.

In electrocardiograms exhibiting low Q.R.S. voltages in the standard leads, the maximum voltages less than 5 mm.:—As a general thesis I would account these as not significant, provided the T. deflections were of reasonable voltage in the standard leads and Q.R.S. deflections and the T. deflections entirely normal in the unipolar chest leads. It must be remembered that each of the standard leads is a synthesis of the rapidly fluctuating voltages in the limbs of derivation and that negativity of the one cancels out negativity of the other, and vice versa. I am inclined to argue that this 'cancelling out' process may be excessive in perfectly normal hearts and that normality of the T. deflections in the standard leads and of the Q.R.S. and T. deflections in the chest leads should more or less completely nullify the presumed adverse significance of the low Q.R.S. voltages in the standard leads. This appears

to be reasonable on the basis of theory, and my belief is that it is acceptable on the basis of clinical observation. The standard leads being synthetic, each lead a mixture of electrical components from two limbs, they are not to be given equality of status with the unipolar chest leads. Unipolar leads are more or less 'pure', each being a fairly accurate portrayal of the E. M. F.'s on that aspect of the heart closest to the chest electrode. On theoretical grounds such leads must be accounted as more likely to be consistent with what is happening in the heart. Notwithstanding the generalization just made, I am not prepared to defend low Q.R.S. voltages in the standard leads to any extreme degree.

I would add that in interpreting chest leads one must keep clearly in mind that the value of the chest lead is inversely proportional to the nearness of the point of application to the surface of the heart. Accordingly, leads from positions 4, 5 and 6 may be vitiated by the heart not approximating the chest wall at those points. For instance, with a small vertically placed heart, No. 6 position (in the axilla) will be a long way from the surface of the heart and the E. M. F.'s will be low and not of much value. On the other hand, with a transversely placed heart, particularly if it is enlarged, the No. 6 position may be expected to give fairly accurate portrayals of the E. M. F.'s on the left ventricle and, accordingly, in such patients this lead is of very great value. Another factor is the thickness of the parietes. In a fat female with a large mamma, graphs made from positions 4, 5 and 6 would be more or less unreliable.

CHAIRMAN ROBINSON—I have asked Dr. H. E. Ungerleider, Associate Medical Director of the Equitable Life Assurance Society to continue with this discussion. Dr. Ungerleider!

DR. HARRY E. UNGERLEIDER—Dr. Birchard through Dr. Robinson sent me a summary of the criteria that he proposed, probably just recently, but it seemed to me so excellent, so well conceived, I think I ought to read it to you, although it

isn't part of my discussion and I did not intend to read it. He states as follows:

- "(a) Q.R.S. voltages of 4 mm. to 5 mm., no unipolar chest leads in the record:—Postpone pending receipt of record showing three standard leads and three unipolar chest leads.
- (b) Q.R.S. voltages of 4 mm. to 5 mm. in the standard leads, and flat T deflections in the same leads, no chest leads in the record:—Decline
- (c) Q.R.S. voltages of 4 mm. to 5 mm. in the standard leads with normal T deflections and three normal unipolar chest leads:—Accept without question.
- (d) Q.R.S. voltages of 4 mm. to 5 mm. in the standard leads, flat T deflections in Leads C.F. 2, or C.F. 4 (or, V-2 or V-4):—Decline

If two of the above mentioned leads be normal and Lead C.F. 6 (or, V-6) depart from normality by having a flat T deflection, I would be inclined to classify the risk as insurable, but 'substandard'. As a guess I would rate at, plus 40 to plus 50.

- (e) With Q.R.S. voltages of under 4 mm. it is unlikely that the other attributes of the graphs which have been mentioned above will approximate normal and I should say that as a general proposition these cases should be declined.

In taking account of the size of the Q.R.S. deflections in the standard leads and also in the chest leads, one should not lose sight of the fact that females tend to have lower electrocardiographic voltages than males and, of course, fat persons lower than those with relatively thin chest walls. Other factors too, and not intrinsic to the heart and about which little is known, affect the size of the deflection."

I commend that classification to you now. My particular discussion of this will be extremely brief.

I agree with Dr. Birchard that low voltage of the Q.R.S. calls for careful scrutiny and that additional electrocardio-

graphic information such as is derived from serial precordial leads is of great value. There has been an increasing tendency to minimize the significance of low voltage of the Q.R.S. complex, particularly when the voltage is only moderately low, i.e., 5 to 7 mm. Very low voltage below 4 mm., as Dr. Birchard indicates, cannot be disregarded. Such extreme low voltage almost certainly denotes heart disease.

It is worth noting that low voltage of the Q.R.S. can be due to a number of factors which we may mention. Among these are:

- Myocardial disease
- Myxedema
- Pericardial effusion
- Chronic adhesive pericarditis
- Emphysema
- Pneumothorax
- Pleural effusion
- Edema of the chest wall, and anasarca
- Scleroderma
- Obesity

It is evident, therefore, that although low voltage of the Q.R.S. complex of 4 to 5 mm. admittedly may occur in normal individuals such a finding dictates careful clinical, and not only additional electrocardiographic, scrutiny. Any of the above abnormalities is a serious impairment. Since true low voltage of the ventricular complex in the limb leads is a rare finding in normal individuals, I do not believe we can lightly dismiss this finding. A careful history and report of physical findings, X-ray of the chest certainly, as well as the additional electrocardiographic studies, as suggested by Dr. Birchard, should be available before we dismiss low voltage of the Q.R.S. in any individual as a normal variant.

CHAIRMAN ROBINSON—I have asked Dr. Bradshaw, Medical Director of the Mutual Life Insurance Company of New York to continue and conclude the discussion on this question. Dr. Bradshaw!

DR. WILLIAM M. BRADSHAW—The occurrence of a QRS of less than 5 mm. in all limb leads, as a solitary finding in an otherwise *normal* electrocardiogram is not commonly seen. However, when it does occur, it makes us wonder about its significance.

Recent studies and opinions by cardiologists now recognize that a low voltage QRS is sometimes seen as a normal variation in routine electrocardiograms of healthy persons. At times, it is the result of slight degrees of right and left axis deviation, where the R and S waves tend to equal each other in Lead 2. It is also recognized that the amplitude of the QRS may be influenced by certain physiological factors such as respiration and changes in body position. Five authorities interpreting electrocardiograms define the minimal normal QRS measurement as 4, 5, 4, 7, and 5 mm. respectively.

Two studies of healthy normal adults in the Army and Navy throw light on this subject:

Among one group of 500 men, the tracing of one individual was found to have QRS waves of less than 5 mm. in all three leads. (1)

Another study of the tracings of 1,000 normal healthy young adults showed 16 instances where the greatest amplitude of the QRS in any standard lead was 5 mm. or less. (2)

The commonest pathologic conditions in which low voltage does occur include myocardial damage resulting from coronary heart disease, congestive heart failure from any cause, myxedema or cretinism, acute or chronic pericarditis, some acute febrile diseases, malnutrition, and clinically recognized vitamin deficiency diseases. Fortunately, in each of these conditions there are usually associated electrocardiographic changes of the nature of ST elevations or depressions, or flat, diphasic or inverted T waves.

The occurrence of low voltage QRS in disease states, without the above associated changes, is unusual, but it may occur. In this event, it is necessary either to obtain a significant

clinical history or previous serial electrocardiograms showing that the voltage had been higher but has decreased, before accepting this variation as a diagnostic sign of disease.

Conclusions:

1. Low voltage QRS in an otherwise normal electrocardiogram is at most suspicious, but not in itself diagnostic.
2. In a normal healthy individual, in whom a reliable history and a careful physical examination fail to disclose any finding of present or past disease, low voltage QRS may be accepted as a normal variation.
3. If there is anything in the history or examination which discloses findings suspicious of heart disease, then low voltage QRS must be accepted as corroboratory evidence.

Insurance Action:

1. If the proof is satisfactory that the low QRS is merely a variation of an otherwise normal electrocardiogram, the applicant otherwise being normal in all respects, acceptance at standard rates is recommended.
 2. If the low voltage is a manifestation of a ratable disease, such as hypothyroidism or malnutrition, the rating should be for that disease only.
 3. If the electrocardiographic finding is a manifestation of some form of heart disease, a declination is indicated.
- (1) Quotation from "A detailed Analysis of the Electrocardiograms of 500 RCAF Aircrew" — Stewart, C. B. and Manning, G. W.: *Am. Heart J.* 27:502-523 — Apr. 1944.
- (2) Analysis of the Electrocardiograms obtained from 100 Healthy Aviators — Graybiel, A., et al: *Am. Heart J.* 27:524-549 — Apr. 1944.

CHAIRMAN ROBINSON—The next question which I thought might be of general interest to our membership was, "*What Constitutes More Than One Attack of Duodenal Ulcer?*" I think we have all had the experience of having an applicant or his physician say that this was a simple, uncomplicated single attack of duodenal ulcer, and then have the company decide that he had two attacks. I hope that, perhaps, we might clarify it,

and I have asked Dr. Rollins, who is Medical Director of the Connecticut Mutual to open the discussion on this question. Dr. Rollins!

DR. HENRY B. ROLLINS—Dr. Robinson's question, "What constitutes more than one attack of duodenal ulcer?" is a pertinent and timely one, first, because of the marked frequency with which we are encountering this problem and, second, because of the multiplicity of factors involved in properly evaluating the history. No discussion is necessary in cases involving (1) hemorrhage, (2) perforation, (3) severe, persistent gastric symptoms, or (4) where there is a crater or a tender and deformed duodenal cap found by combined X-ray and physical examination. The less severe forms of symptomatology (hyperacidity, soreness in the epigastrium, spasm, abdominal distress, dyspepsia, etc.) with borderline or negative X-ray findings present our real problem, particularly so if the attending physician comments that the condition was of little significance.

It is this group that interests us most in determining the answer to the question: Are we merely dealing with a continuance of the original duodenal complex, which is entirely a question of degree, severity, duration, etc., or an actual lighting up of a chronic process?

What about mildly recurring episodes of epigastric distress relieved by self-imposed dietary restriction and medication? Our textbooks speak of the seasonal exacerbations (spring and fall) with relation to peptic ulcer. Are these to be considered as second attacks? This type of history is quite typical of the majority of ulcer patients if carefully questioned, but it is not considered a recurrence either by the laity or by the profession generally.

How about the check-up or follow-up gastro-intestinal X-ray examinations? In numerous instances these are prompted by recurrent symptoms, minimized, however, both by the applicant and by the physician. If the X-ray is now negative,

what are we to do? What about the chronically deformed duodenal bulb on X-ray examination in the absence of any recurrent symptoms?

In attempting to determine what constitutes a second attack, it appears to us that the whole evolution of the ulcer state should be considered rather than the ulcer itself. Today we are stressing the constitutional and psychosomatic aspects of this malady. If this is true, it is by definition a general disorder with a tendency to recurrence and chronicity. As one internist aptly stated, the duodenum is the site of the disease but not the seat of the disease. Secondly, we attempt to secure an accurate history. What were the severity and duration of symptoms? Not every mild digestive upset following a dietary indiscretion is worthy of being called a second attack, nor do we believe that the patient who avoids spicy and greasy foods is necessarily under treatment. Many patients are advised to continue indefinitely the bedtime pill or powder, and we consider this good medical advice. Thirdly, we try to evaluate the type of patient with whom we are dealing. We are all familiar with the fact that severity of symptomatology is not necessarily related to severity of ulcer, and vice versa. How many times have we taken histories profoundly typical of ulcer only to be dismayed by negative X-ray reports? Is this nervous indigestion or hyperacidity? On the other extreme, we have all seen the lumberjack, brought in, in extremis from hemorrhage or perforation, who later boasts of his "iron stomach" and denies any history of indigestion. Obviously these wide variations in subjective symptomatology are not related to the ulcer, *per se*, but rather to other factors, not the least of which is the sensitivity of the individual. The personality make-up should be evaluated in attempting to assay the severity or significance of the symptoms. Lastly, we must take into consideration the background and training of the attending physician and his qualifications for X-ray diagnosis. Accurate interpretation of fluoroscopic and X-ray examination of the gastro-intestinal tract

requires the utmost skill. The variability in successive X-ray diagnoses on many of these cases cannot be properly explained by the clinical course of the disease, but must be attributed to the variable skill of the operator and technical difficulties. Witness also the wide divergence in opinion in the profession itself as to what constitutes X-ray evidence of ulcer. One school of thought believe that any persistent deformity of the bulb is evidence of ulcer, while the more conservative group hold that the crater of an ulcer must be demonstrated by the barium fleck.

In conclusion, we regret that we have no mathematical formula or strict set of rules to govern what constitutes a second attack of ulcer. We do attempt to analyze each case carefully from the standpoint of these four variable factors. In some cases we believe a diagnosis of a second attack is justified on simply a history of self-imposed regime of diet and medication, if such regime was prompted by typical ulcer symptoms of sufficient severity and duration. I think we should be careful to protect the hypersensitive, conscientious individual who, by meticulous analysis of his symptoms, makes his case appear unduly severe. More often, however, we must be wary of an understatement of the facts, consciously or unconsciously, or by the agent's prompting. The X-ray report is correlated with medical history and not necessarily accepted as the final judgment, but, rather, evaluated with relation to the skill of the operator.

CHAIRMAN ROBINSON—We will ask Dr. Roland A. Behrman, Medical Director of the John Hancock Life Insurance Company of Boston to comment further on this question. Dr. Behrman!

DR. ROLAND A. BEHRMAN—When clinical opinion concerning etiology and cure of peptic ulcer is as uncertain and controversial as is now the case, it is not to be wondered at that we Medical Directors have no simple solution to the problem posed by the topic, "*What constitutes more than one attack of duodenal ulcer?*"

DUODENAL ULCER

Without Operation

One attack

Simple attack, acute, short duration, no hemorrhage

Limit \$25,000 0 to 6 months

0 to 7 yrs. 7 months to 1 year

Limit \$50,000 1 to 2 years

8 yrs. and 2 to 5 years

after 5 years and after

RNA

1 extra \$10 per M

1 extra \$2.50 per M

0 to 20

Standard

AI

WP

No
Yes
Yes
YesNo
No
No
Yes

Two attacks, or history of hemorrhage, or treated surgically (gastro-enterostomy)

0 to 1 year

1 to 2 years

2 to 3 years

3 to 4 years

4 to 5 years

5 years and after

RNA

100

75

50

25

0 to 20

No
No
No
Yes
YesNo
No
No
No
Quest.

More than 2 attacks

0 to 4 years

4 to 5 years

5 to 6 years

6 to 7 years

7 to 8 years

8 years and after

RNA

100

75

50

25

0 to 20

No
No
No
No
Yes
YesNo
No
No
No
Quest.

<i>With Operation</i>			
More than one operation		No	No
Decline except if second operation was a sub-total gastrectomy -- treat as sub-total gastrectomy		No	No
History of repeated hemorrhages	RNA	No	No
Recurrence following operation		No	No
Decline under 4 years		No	Yes
After 4 years -- treat as "More than 2 attacks"			
Duodenal or gastric ulcer, treated surgically (sub-total gastrectomy)			
Limit \$25,000 Ulcer Form 489 required from attending physician	RNA		
0 to 1 year	100		
1 to 2 years	65		
2 to 3 years	35		
3 to 4 years	25		
4 years and after			
Recurrences following sub-total gastrectomy	RNA		

Duodenal ulcers fall somewhat arbitrarily into one of three categories:

First, those that are acute, definitely demonstrable by X-ray, promptly responsive to medical and dietary treatment and not subject to recurrence. These constitute perhaps 10–20% of all cases and are probably better recognized as a group in life insurance medicine than by clinicians since, as patients, they respond as in any acute illness.

Second, those which respond slowly but progressively to treatment of months' or 2 or 3 years' duration and eventually a seeming cure.

Third, those which might be labeled "chronic" where symptoms persist—with remissions—for years, ameliorated while on strict treatment but flaring up with lapse in treatment or on exposure to disturbing psychic factors or perhaps without recognizable cause. In contradistinction to the acute type of ulcer mentioned in Group 1, ulcers in Groups 2 and 3 are the ones so often seen in clinics and which probably account for clinical pessimism as regards a permanent cure.

In any of these groups, where an ulcer has been found by X-ray or strongly suspected even without roentgenological evidence and where there has been complete freedom from symptoms for a period of two years or more following cessation of all but simple dietary precautions, we can assume that a temporary cure has been effected. If, after this period, there is a recurrence of suspicious symptoms, it seems reasonable to us to regard this as a second attack, recognizing, however, that it might be merely a recrudescence of the former ulcer but, in our opinion, nevertheless indicating that the case should carry a heavier substandard rating.

CHAIRMAN ROBINSON—To conclude the discussion on this topic, I have asked Dr. Howard B. Brown, Associate Medical Director of the Massachusetts Mutual to comment. Dr. Brown!

DR. HOWARD B. BROWN—The diagnosis of recurrent duodenal ulcer is simplified, of course, when there is primary or secondary X-ray evidence or a history of hemorrhage. In requesting a brief discussion, I assume that the Chairman was most concerned with the cases which do not present positive evidence.

Under these circumstances, I feel that we must rely more upon the history obtained from the attending physician than that secured from the applicant. The important feature is the appearance of discomfort whenever gastric acidity is not neutralized. There may be localized tenderness at these times. Usually, the pain is severe enough at some period in the cycle to awaken the patient at one- or two-hour intervals during the night.

When there is an extended history, but no clear evidence of recurrence, we often rate empirically on a two attack basis. These people may have chronic duodenitis with occasional superficial ulcerations.

Spastic colitis does not present a difficult problem in differential diagnosis when accurate history is available, inasmuch as the discomfort in a majority of cases is not rhythmic nor well localized.

CHAIRMAN ROBINSON—Our last subject is Diverticulosis and Diverticulitis. I have asked Dr. F. W. Rolph, who is Associate Medical Director of the Confederation Life and an outstanding gastro-enterologist in the City of Toronto, to open the discussion on this subject. Dr. Rolph!

DR. FRED W. ROLPH—From both the clinical and insurance standpoints the important location for these lesions is in the colon, and more especially the sigmoid colon. Diverticula are herniations of the mucous membrane through gaps in the muscularis; symptomless in themselves they are apt to fill with stagnating intestinal contents, with resulting ulceration and inflammation. They are of rare occurrence under the age of forty, but in later years may be found in about twelve per cent of the population.

Diverticulitis very commonly shows itself in three stages:

- (1) With colonic spasm—
Most cases do not extend beyond this, but about twelve per cent go on to
- (2) Infiltration or
- (3) Perforation and abscess.

The mortality in these severe forms is high, probably ten per cent.

The diagnosis between diverticulitis and carcinoma is difficult, but under the age of forty an obstruction is much more likely to be due to malignancy, and in older ages cancer of the sigmoid is about three times as prevalent as an obstructing diverticulitis.

For insurance purposes, I believe we may usually disregard diverticula, which are not very numerous or large, have not caused symptoms and have been discovered in a routine X-ray, but the milder types of diverticulitis with vague symptoms resulting from intestinal spasm, should not be considered on a standard plan.

Finally we must consider the insurability of individuals who have had one, or more, severe attacks of diverticulitis, and who have been treated either medically or surgically for the condition. In my experience these attacks are extremely likely to recur and constitute an ever present source of danger. I believe that the very shortest waiting period to be considered would be two years and that any policy issued must be quite heavily rated.

Other types of diverticula I shall consider very briefly:

- (1) Duodenal.

There are two main types:

- (a) Congenital—occurring in the 2nd and 3rd portions and not of much clinical importance unless situated at the duodeno-jejunal juncture, in which situation acute obstruction may occur.
- (b) Ulcer diverticula, usually in the first portion and to be considered as duodenal ulceration.

(2) Jejunal.

These are of rare occurrence but may give rise to severe symptoms.

(3) Meckel's Diverticulum.

This is caused by the failure of the vitello-intestinal duct to close and atrophy. It may be the site of inflammation causing intestinal obstruction and in children not infrequently gives rise to peptic ulceration and hemorrhage.

CHAIRMAN ROBINSON—Discussion of this subject will be continued by Dr. Ernest Dewees, Medical Director of the Provident Mutual of Philadelphia. Dr. Dewees!

DR. ERNEST J. DEWEES—I just want to call your attention to the review of this subject in 1941 by Dr. E. S. Williams, one of our own members, and I would suggest that, any of those who wish to pursue the subject further, that they refresh their memory in relation to this review of the literature and this paper which was so well presented at that time. So much for that.

The former speaker has discussed with you the conditions that are confronted in this disease. I will only indicate to you then what is our usual practice in my own company in the handling of these cases. So far as I am aware, we have no satisfactory figures; no satisfactory criteria on which to base a satisfactory rating schedule. This means we must rely largely upon clinical experience and the reports which come from various clinics of the country. This also further means that we will find great variation among the various medical departments of the country concerning the handling of this particular condition.

In the Provident Mutual we are inclined to divide these cases into three groups: Diverticulosis which is known to be present under forty years of age, where there are no gastrointestinal signs and never have been that would be related to this condition, where in every respect the individual is healthy

and well and has a clear history—we consider with a moderate rating, that's all. Diverticulosis cases are necessarily congenital ones, referred to by a former speaker. Where there is proven diverticulosis we take a conservative point of view and give a small rating. That would likely be for most of us our first or lowest rating. Secondly, those in whom there has never been a definite diagnosis of diverticulosis, but those who have indefinite signs of a digestive disturbance or health complaints of one sort or another, we consider as questionable and rate as high as plus 50. Cases with a history of diverticulosis any time in the past or who may have a disturbance at the present time are declined by the Provident Mutual.

CHAIRMAN ROBINSON—The discussion will be concluded by Dr. Albert S. Irving, Medical Director of the Commonwealth Life. Dr. Irving!

DR. ALBERT S. IRVING—A study of the literature indicates that the subject of diverticulosis and diverticulitis is of surprising interest to the life insurance medical director—surprising in that the contribution to our knowledge of the subject consists of a paper presented to our organization on October 22, 1925 by John F. Erdmann and a paper given before the Medical Section of the American Life Convention by Dr. Ennion S. Williams, Medical Director of The Life Insurance Company of Virginia, one of our members, on June 19, 1941. There are comparatively few other contributions—most of these have been given by the surgeons and the statistics do not cover many cases. One paper is presented in the Journal of The American Medical Association for the week of October 19, 1946, this past week.

Diverticulosis is a condition in which saccular pouches protrude from the lumen into the wall of a portion of the digestive tract. They are found in all areas from the esophagus to the rectum but the term is in practice considered as referring primarily to the colon, chiefly of the sigmoid flexure and secondarily of the caecum.

This condition causes no symptoms and is important in that, because of its presence, it offers an opportunity for irritation or infection to cause an inflammation of the diverticulum which condition is known as diverticulitis.

About 1% of all X-ray examinations of the gastro-intestinal tract show the presence of diverticula but the incidence above age 40 is from 5% to 10% and it is thought from 12% to 20% of these will develop diverticulitis.

Diverticulitis is a condition which is found localized most frequently in the lower left quadrant, involving males from 40 to 50, about 4 to 5 times as often as females, and in individuals who are short, heavy, and overweight in build—children are occasionally affected.

The pathology is the same as in appendicitis ranging from an acute catarrhal attack to a gangrenous condition and it may be self limiting by the formation of adhesions which may even seal off a perforation. This is understandable when you realize that from one to the three layers of the bowel may be involved, mucous membrane, muscularis, and serosa.

Surgical treatment is called for in cases with perforation complicated by abscess formation or generalized peritonitis, in cases of obstruction and in cases when a fistula develops involving bladder, large or small intestine, appendix, uterus, etc. The result of surgery in acute cases is fairly good but the chronic cases, especially those requiring gut resection, present a high immediate mortality 10% and up—Erdmann reports a 4% recurrence and that must be taken into consideration in underwriting these cases.

Diverticulosis, found incidental to other examination, which has caused no symptoms, is apparently not enough to justify rating and probably is satisfactory at standard rates.

Following operation, cases probably should be postponed for two years because of the possibility of recurrence and because there is evidence statistically to indicate other areas of the gastro-intestinal tract are probably involved too—after

two years these cases can be held at substandard rates for 10 years ranging from Table 1 to Table 4, 125% to 200% of mortality.

The cases with complications, particularly a wide-spread peritonitis and following wide-spread adhesions or a perforation, should be rejected.

There is one additional comment I would like to make and that is several of the surgeons have made the statement that the appendix itself is diverticular, but the condition we are discussing now consists of numerous such areas and, therefore, ratings should be much more severe than we would impose for appendicitis. We are concerned with a condition which is effective as far as mortality is concerned, not morbidity.

....President Streight resumed the Chair....

PRESIDENT STREIGHT—In concluding our formal program on behalf of the Association, let me offer our warmest thanks to our guests and members and all others who took part in the program. I wish to offer my sincere thanks to those who so ably assisted me throughout the year, particularly the members of the various committees who have devoted much time and thought to the affairs of the Association.

I wish to thank our retiring secretary, Dr. Edwin G. Dewis, for his loyalty and unfailing support on every occasion. With his help every difficulty was resolved.

Looking to the future, our meetings will reflect the interest and effort we put into them. Your officers require your support in every undertaking throughout the year. Let each of us when asked to help answer the call and do our part towards making these meetings the interesting and informative gatherings we wish them to be.

One other thing, I would like to call your attention to the fact that during the period from 1942 to 1945 the business affairs of this Association were carried out by Dr. William

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Bolt, a most worthy man who devoted a great deal of time and attention to various things without having the benefit of an annual meeting to encourage and support him. I would like to have someone suggest that we offer him a vote of thanks for the very able way in which he did conduct the affairs of the Association during that period.

It was so moved, seconded and unanimously carried amid applause.

PRESIDENT STREIGHT—My only remaining duty is the very pleasant one of introducing my successor. May I bespeak for him the same co-operation and courtesy which you so generously have extended to me. Dr. Jimenis! (Applause.)

DR. ALBERT O. JIMENIS—Well, there are only a few of us left, and I wish to say first of all that I highly appreciate this honor and I will do my best to provide a meeting next year which may closely approximate the success attained this year by Dr. Streight. The changes that we made yesterday in our Constitution and By-Laws should now give us more time for mortality investigations.

In view of the changes that we heard of yesterday, the administration of one of our Departments is going to have a change, and Mr. Valentine O. Powell, Vice-President of the Prudential, who was Chairman of that Committee, has asked me to remind you that we will need an Executive Secretary. The Joint Committee, which will run this Department, has not yet been formed and we cannot say certainly that the Executive Secretary will be a doctor. We hope he will be. In the meantime if any of you know of any individual who might be qualified for such work just send us a memorandum so that we may have his name and address at hand when we need it—either to me or to Dr. Hutchinson, Chairman of the Medical Section of the American Life Convention.

Gentlemen, is there any further business to come before this meeting?

DR. ROWLEY—I am happy to have the privilege and honor of being the first to formally address you as the new occupant of the high office of President of this Association. You are all of one mind, I am sure, in feeling that this has been one of the most interesting and profitable meetings of the Association that we have been privileged to attend. There are some new faces among us, and in the coming years the younger members will be in a position to contribute to the knowledge that will be needed.

I would like to move that this Association pass a hearty vote of thanks to Dr. Streight.

The motion was duly put, seconded and unanimously passed.

DR. STREIGHT—Thank you very much.

* * *

A motion to adjourn was made, seconded and carried, and the meeting was adjourned.

* * *

The following members, delegates and guests were present at some time during the sessions: Doctors J. W. Abbott, E. H. Allen, G. E. Allen, W. J. Allison, H. H. Amiral, K. W. Anderson, T. D. Archibald, E. M. Armstrong, T. M. Armstrong, W. B. Aten, H. A. Bancel, G. H. Barber, N. J. Barker, J. R. Beard, E. W. Beckwith, J. E. Bee, R. A. Behrman, M. B. Bender, R. W. Benton, P. E. Bernstein, C. H. Best, J. R. Biggs, C. C. Birchard, W. R. Bishop, J. E. Boland, William Bolt, E. C. Bonnett, J. T. Bowman, W. M. Bradshaw, K. F. Brandon, A. W. Bromer, — Brooks, C. T. Brown, F. R. Brown, H. B. Brown, E. R. Bush, E. J. Campbell, H. B. Campbell, F. H. Carber, P. E. Carlisle, D. W. Carter, Jr., J. P. Chapman, E. D. Chesebro, C. P. Clark, M. H. Clifford, B. R. Comeau, F. R. Congdon, F. V. Costello, D. B. Cragin, B. B. Crohn, F. P. Cross, Simon Dack, R. M. Daley, W. L. Davis, H. D. Delamere, E. J. Dewees, E. G. Dewis, F. R.

Dieuauide, E. S. Dillon, H. W. Dingman, A. H. Domm, W. W. Dow, J. T. Eads, T. M. Ebers, L. B. Ellis, J. L. Evans, A. H. Faber, J. G. Falconer, R. K. Farnham, H. H. Fellows, W. E. Ferguson, R. M. Filson, R. W. Finegan, V. J. Fingar, Frederick Fink, E. M. Freeland, F. I. Ganot, D. S. Garner, J. T. Geiger, W. M. Genthner, E. E. Getman, J. M. Gilchrist, R. A. Goodell, H. E. Goos, R. J. Graves, H. M. Gray, George Greenway, R. S. Gubner, J. R. Gudger, L. E. Haentzschel, Llewellyn Hall, E. J. Hardin, Frank Harnden, L. E. Hathaway, W. C. Hausheer, H. M. Hawkins, W. D. Heaton, E. M. Henderson, E. V. Higgins, W. L. Hilliard, D. W. Hoare, — Hoffman, C. O. Hollinger, J. C. Horan, J. L. Humphreys, J. H. Humphries, J. E. Hunsinger, J. R. B. Hutchinson, A. S. Irving, J. G. Irving, W. A. Jaquith, A. O. Jimenis, A. E. Johann, H. J. Johnson, J. W. Johnson, T. D. Jones, C. S. Keefer, C. H. Kelley, E. F. Kerby, H. B. Kidd, D. G. Kilgore, H. B. Kirkland, Phillip Lambkin, P. H. Langner, Jr., A. J. Lanza, A. L. Larson, L. H. Lee, E. P. Leeper, J. M. Livingston, O. S. Lloyd, H. C. McAlister, F. M. McChesney, C. B. McCulloch, A. J. McGanity, George McLean, L. J. McLellan, A. R. McMahan, W. J. McNamara, Charles Maertz, S. J. N. Mtgwood, W. L. Mann, J. C. Miller, L. C. Miller, R. C. Montgomery, J. F. Moore, Jr., M. A. Murphy, S. A. Narins, E. C. Noble, Eduard Novak, Jan Nyboer, A. J. Oberlander, Herbert Old, M. I. Olsen, B. H. Olson, G. T. Pack, W. C. Page, C. B. Parker, A. E. Parks, D. S. Pepper, C. A. Peters, C. B. Piper, Cullen Pitt, J. J. Post, R. W. Pratt, O. S. Randall, J. H. Ready, P. V. Reinartz, W. A. Reiter, T. W. Reul, W. M. Reynolds, D. F. Ridders, G. P. Robb, D. C. Roberts, A. J. Robinson, H. B. Rollins, F. W. Rolph, R. C. Roskelley, T. F. Ross, R. L. Rowley, W. W. Rucks, N. R. Ruud, K. F. Schaefer, S. B. Scholz, L. P. Schroeder, B. T. D. Schwarz, E. D. Sherman, Albert Seaton, R. C. Secor, J. T. Sheridan, D. M. Shewbrooks, R. R. Simmons, W. A. Smith, F. L. Springer, H. F. Starr, J. B. Steele, D. F. Steuart, E. M. Stevenson, H. M. Stewart, I. R. Stidger, A. R. Stone, S. J. Streight, E. V. Sweet, L. G. Sykes, Irvin Tabershaw, K. J.

Members, Delegates and Guests Present 307

Thomson, W. E. Thornton, Joseph Travenick, Jr., H. B. Turner, H. E. Ungerleider, B. W. Vale, Euen Van Kleeck, W. R. Ward, C. F. Warren, R. L. Weaver, Jefferson Weed, D. E. W. Wenstrand, S. S. Werth, A. A. Willander, E. S. Williams, A. A. Wills, Jr., A. C. Wilson, G. E. Woodford, L. S. Ylvisaker, and Messrs. L. F. Baker, H. C. Bates, Ray Burke, Leigh Cruess, Valentine Howell, Arthur Hunter, Edward King, G. C. Kingsley, J. A. McLain, L. S. Parker, P. G. Shepherd, O. G. Sherman, P. V. Wells and Miss A. M. Lyle.

Total attendance at all sessions, 237.

MEMORIALS

DR. EMMANUEL P. BENOIT

1869—1946

Dr. Emmanuel P. Benoit was born in Montreal, December 29, 1869. He was admitted to the medical practice in 1892.

In 1899, he was named professor of internal medicine at Laval University and, a few years later, honorary professor of medical clinic at Notre-Dame Hospital, Montreal.

He was called to the Council of the Faculty of Medicine in 1913, and became Secretary of that body in 1929, an office which he held until his death.

His valuable contribution to both the medical science and the medical education brought him a number of enviable distinctions, namely, Officier d'Académie, Corresponding member of La Société Médicale des Hôpitaux de Paris, Member of the College of American Physicians, Member of the Royal College of Canadian Physicians and honorary member of La Société Médicale de Montréal.

Beside taking an active part in hospital and university life, Dr. Benoit has also devoted twenty-five years of his life to the professional interests and medical education of nurses perhaps the most important of his many activities and one which he held at heart.

Dr. Benoit joined La Sauvegarde Life Insurance Company in 1908, as Medical Director.

DR. HAROLD E. BOGART

1896—1943

It is with great regret that the New York Life Insurance Company announces the sudden death of Dr. Harold E. Bogart on December 17, 1943. Dr. Bogart had been an Assistant Medical Director of the Company since June 26, 1935.

He was born at Ithaca, New York on October 31, 1896, the son of Elmer E. and Margaret Bogart, still living. The family moved to New York in 1906 and Dr. Bogart attended the Morris High School, of which his father was Principal, graduating in 1914. He attended Cornell Medical College, receiving an A.B. Degree in 1918 and an M.D. Degree in 1921. He interned at Gouverneur Hospital, New York City.

Dr. Bogart entered the service of the New York Life Insurance Company on December 8, 1924. His ability was soon recognized and he became a Medical Board member on April 1, 1928. He was made Medical Supervisor on December 8, 1931 and was appointed to the rank of Assistant Medical Director in 1935. His untimely death was deeply regretted by all who were associated with him.

DR. CHARLES LEONARD CHRISTIERNIN

1878—1944

Dr. Charles Leonard Christiernin, Medical Director of the Metropolitan Life Insurance Company, died suddenly of a heart attack on October 18, 1944 while on vacation at his camp in the Adirondacks at the age of 66. Entering the service of the Company as a Medical Examiner in Boston, Massachusetts, he joined the Medical Staff in the Home Office in April 1911. In 1916 he was made Assistant Medical Director and in 1935 was appointed Medical Director in full charge of the Company's medical work, and although past the retirement age he elected to continue his duties because of the absence on military duty of a large part of his medical staff. A modest, unassuming man, his manner nowise suggested his accomplishments. He carried on his work with a consideration for others that won him the friendship of all with whom he had dealings.

In the Medical Directors Association he was for many years an active member. He was Treasurer of the Association from 1918 to 1929, Vice President from 1929 to 1931 and

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President from 1931 to 1932. He was a member of the Council and a member of the M.I. B. Committee from 1940 to his death.

Dr. Christiernin was born in Boston, Massachusetts on February 10, 1878, the son of Henry P. and Rosalie Christiernin.

He attended Boston Latin School and, later, Harvard, receiving his degree of A.B. from Harvard College in 1902, and his degree of M.D. from Harvard Medical School in 1906.

He is survived by his wife, Mrs. Regina Scott Hall Christiernin and one son, First Lieutenant Charles Leonard Christiernin, Jr. To them goes the abiding sympathy of all of us who knew him.

DR. HAMILTON CHALMERS CRUIKSHANK

B.A., M.B., D.P.H.

1888—1942

Dr. Hamilton C. Cruikshank of Toronto, Ontario, died following a short illness at his home on Christmas day 1942, from coronary disease.

Born in Hamilton, Ontario, on August 30th, 1888, Dr. Cruikshank was educated at Hamilton schools and at University College, University of Toronto, where he completed the Arts course in 1909. After four years of medical studies at the same University he entered the medical service of the Canadian Pacific Railway, and was in British Columbia for two years. At the beginning of the Great War, Dr. Cruikshank enlisted with the Canadian Army Medical Corps and was in France during 1915, 1916 and 1917, attaining the rank of Captain. In 1917-18 he was in charge of serology, Base Laboratory M. D. No. 2.

On his return home he entered the Department of Soldiers Civil Re-establishment, where, in 1919 and 1920 he was in charge of serology in conjunction with the Connaught Laboratories and the Department of Preventive Medicine, University

of Toronto. He graduated in Medicine from the University of Toronto in 1919 and received his diploma in Public Health two years later. In May 1920 he entered the Department of Public Health as Director of Laboratories and in September 1924 he was promoted to the position of Deputy Medical Officer of Health for the City of Toronto where his work against smallpox and other communicable diseases won him high praise. He occupied this post with distinction until his appointment as Medical Officer for the Manufacturers Life Insurance Company on January 1st, 1927.

Dr. Cruikshank was a member of the Association of Life Insurance Directors of America and of the Medical Section of the American Life Convention, as well as the Medico-Actuarial and Public Health Committees of the Canadian Life Officers Association.

His kindly and genial personality made him a great favorite, not only with his fellow executives and Head Office associates, but also with the Field Force of the Company generally to whom he was well known. He will be greatly missed by all who knew him.

DR. JOSEPH E. ENGELSON

1882-1946

Dr. Engelson, after an illness lasting one year, died at his home in Plainfield, N. J. on March 17, 1946. He had been a member of this Association since 1928 and had been associated with the Mutual Life Insurance Company of New York since 1927.

Born in New York, he attended schools in this city and was graduated in medicine from Columbia University, the College of Physicians and Surgeons, in 1906. For the next two years he completed his internship at Roosevelt Hospital and immediately took postgraduate training at the Sloane Hospital for Women. He entered private practice in 1910, specializing in gynecology and obstetrics. He remained active in that specialty until joining the Mutual Life in 1927.

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During World War I he was captain in the Medical Corps and was a member of a surgical team made up of Roosevelt Hospital alumni. This unit saw extremely active service in the war zone in France.

By close association of many years, his associates recognized in him a faithful and dependable friend. His loyalty to the company and his desire to bear his full share of the work were among his outstanding attributes.

He is survived by his wife, Grace M. Engelson, and one son.

DR. FRANCIS CHARLES EVERS

1893-1945

Dr. Evers died at his home in Mount Vernon, New York, on February 17, 1945, due to a heart condition from which he had suffered for a period of approximately five years.

He was born August 30, 1893 in Mount Vernon, New York. After completing his preliminary studies he matriculated at Fordham University, from which he received the degree of Doctor of Medicine in 1917. Following an internship at the Fordham Hospital, he entered the Medical Corps of the United States Navy, in which he served for four and one half years.

Upon resigning his commission in the Navy, he became an examiner for the New York Life Insurance Company and shortly thereafter, on January 1, 1922, joined the Company on a full time basis as a member of the Medical Board.

His aptitude, ability and general understanding of the problems of insurance medicine earned for him promotion to the post of Assistant Medical Director in 1930. In 1934, he was made a Medical Director and fulfilled his duties with the Company in this capacity until his death.

He is survived by his wife, one son and two daughters.

DR. WILLIAM GUSTAV EXTON

1876-1943

Dr. William Gustav Exton, born at Savannah, Ga., February 25, 1876, died at Mt. Sinai Hospital in New York on March 12, 1943. His early education was directed by private tutors, later he attended Peekskill Military Academy, received the degree of Bachelor of Arts at Columbia University and graduated as a Doctor of Medicine from that institution in 1896. From then until 1899 he was house physician at Mt. Sinai Hospital and during the two years which followed was engaged in postgraduate and research work at the Pathological Institute in Vienna and in London and Paris. Following five years of general practice in New York City he devoted the period, 1907 to 1916 to special practice, teaching and research in urology.

In 1914 he became associated with The Prudential Insurance Company and until his death was Director of the Laboratory and Longevity Service of that Company.

From 1900 to 1941 he published 50 papers describing the results of his original work and investigation. Eight of these appeared in the Proceedings of this Association. His early writings had to do with bacteriological subjects, his special interest in urology was reflected in his writings during the following period and after 1920 his contributions to the literature were related mostly to laboratory methods. The subjects included those dealing with the qualitative and quantitative determination of the proteins and sugars as found in the urine, blood analyses as related to sugar, hemoglobin, red cell count, fibrinogen as an index of disease, iron, cholesterol, acidosis and alkalosis and the significance of the plasma carbon dioxide capacity, and renal and glycosuric functional tests. His writings also included description of laboratory instruments of his own invention, notably the euscope and the photoelectric scopometer. Other publications evidenced his keen interest in pre-clinical medicine and his thoughts on periodic health reviews.

He was president of the American Society of Clinical Pathologists, 1926-1927, and a member of the American Urological Society, The Optical Society of America, The New York Pathological Society, The American Chemical Society and the American Medical Association, and recipient of the Burdick Memorial Medal of the American Society of Clinical Pathologists.

Endowed with a brilliant mind and an altruistic nature he created a loyal group of associates and friends. In 1905 he was married to Florence Phillips of Atlanta, Ga., who survives him, as do three sons all officers in the United States Navy.

DR. ROBERT HEWITT FELDT

1908-1945

Doctor Robert Hewitt Feldt, Assistant Medical Director of The Northwestern Mutual Life Insurance Company, died in Milwaukee on the eighth of October, 1945. He had suffered with a valvular disease of the heart for some years, but had the courage to continue at his desk with considerable regularity until the last ten days of his life.

Born in Monmouth, Illinois, November 27, 1908, Dr. Feldt received his early education in the Monmouth schools and Monmouth College. Upon graduation he entered the Northwestern University Medical School, in which he made an outstanding record, graduating in the upper tenth of his class. He served as an interne in Wesley Memorial Hospital in Chicago for six months and in Cook County Hospital for seventeen months.

Dr. Feldt's excellent training and natural ability fitted him exceptionally for the work of the Medical Department of The Northwestern Mutual, which he joined June 1, 1935. His untimely death cut short a most promising career in his chosen profession. His judgment was always sound and he constantly gave evidence of co-operation and aptitude for developing statistical studies. He had a deep sense of responsibility and

showed unfailing courtesy and consideration for others. All his associates sincerely liked and respected him.

Outside of business hours Dr. Feldt was active in the cardiac clinic of the Milwaukee Children's Hospital. In 1943 he was awarded the Milwaukee Academy of Medicine's annual prize to recent graduates for his treatise, "Sulfanilamide As A Prophylactic Measure In Recurrent Rheumatic Infection." This was based on a controlled study involving 131 "patient-seasons" from this clinic and was published in the American Journal of Medical Sciences, April, 1944. Other articles relating to blood pressure studies had appeared earlier in the same journal and in the American Heart Journal.

As an avocation Dr. Feldt also contributed many articles on medical subjects to national lay periodicals. Nine of these appeared in The American Mercury and others in such magazines as Reader's Digest, Liberty, Canada Digest, Scientific American and Science Digest. He was a member of the Authors' League of America.

He is survived by his wife, two sons, and his mother.

DR. ROBERT ALEXANDER FRASER

1878-1945

Dr. Robert Alexander Fraser, formerly Chief Medical Director of the New York Life Insurance Company, died at Greenwich, Connecticut on Thursday, November 1, 1945.

Dr. Fraser was born in Toronto, Ontario on March 5, 1878. He was educated in Toronto Public Schools and at Harvard College. He received his medical training at Trinity Medical College. Following his graduation, he served as interne in Toronto and Buffalo, N. Y., after which he came to New York City where he practiced from 1907 to 1913. During this period he was connected with the Tuberculosis Clinic of New York Dispensary, New York City Health Department and Bellevue Hospital.

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He served as Lieutenant with the Forty-eighth Highlanders of Canada and later was a Lieutenant in the First Army Medical Corps.

Dr. Fraser first became associated with the New York Life Insurance Company in 1913 following experience in the fields of public health and private practice. His abilities as an administrator and executive, combined with his extensive knowledge of medicine and its relationship to sound life insurance underwriting practices and procedures, were recognized by the company and led to his steady advancement. He was appointed Medical Supervisor in 1917, Assistant Medical Director in 1920, Associate Medical Director in 1927 and in 1934 he was elected Chief Medical Director. In 1942 he was named Chairman of the Medical Committee and a member of the Underwriting Committee. His high standing in his chosen field was recognized by the Association of Life Insurance Medical Directors. He was Editor of the Proceedings of this Association 1929-1932, Vice-President 1932-1933 and President 1934-1935.

In recent years Dr. Fraser had not enjoyed the best of health. However, conscientious devotion to the affairs of the company led him to persist in carrying on his duties with a courage and determination which won for him the admiration of all who knew him, especially those who were closely associated with him. He will be greatly missed by his wide circle of friends in the Association.

DR. WILLIAM WALTER HOBSON

1884-1945

William Walter Hobson was born in Paterson, New Jersey, January 20, 1884, where he received his early education. He died January 4, 1945, of an acute coronary occlusion.

In 1910, he graduated from the College of Physicians and Surgeons at Baltimore, now united with the Medical School of the University of Maryland. He served his internship at the Mercy Hospital, Baltimore. He was a member of the Phi

Chi Medical Fraternity and the American Medical Association. He entered the practice of medicine in Baltimore in 1910, as surgeon for the Montclair Shops of the Baltimore and Ohio Railroad, and also as traveling surgeon for consultation of the entire system.

In 1916, he became associated with the Reliance Life Insurance Company of Pittsburgh as Assistant Medical Director. He was elected Associate Medical Director July 1, 1936, and Medical Director on January 26, 1943. Over many years he was also a Medical Referee for a large number of other insurance companies.

Dr. Hobson was quiet, unassuming, and of a retiring nature, but was none-the-less widely known and very well liked throughout the Company. He was an indefatigable worker student of medicine, and had a keen insight into the problems of insurance medicine.

His sudden death leaves a void in his Company which will be felt for many years.

DR. CHARLES BRUCE IRWIN

1881-1943

Charles Bruce Irwin, Vice President and Medical Director of the North American Life Insurance Company of Chicago, died of a heart ailment while driving to his home on the evening of September 21, 1943. He was born in Westminster, Maryland, April 12, 1881, and graduated from the University of Maryland Medical School in 1904. He practiced medicine in Kansas City, Missouri, until July 1, 1919, at which time he was elected Medical Director of the North American Life. He became a member of the Board of Directors in January 1932 and Vice President and Medical Director in 1939.

In 1912 he was married to Miss Norine O'Brien of Kansas City, Missouri. Surviving him are his widow and two sisters, Mrs. Mary Cunningham and Mrs. Elizabeth Jefferis, both of Westminster, Maryland.

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He was elected a member of The Association of Life Insurance Medical Directors in 1919, and was a regular attendant at its annual Conventions continuously through the years. He was also a member of The American Medical Association and The Chicago Medical Society. He was one of the founders of the Tuberculosis Society of Kansas City, Missouri, and for many years he had been a widely recognized authority on heart diseases. In addition to doing an outstanding job as Medical Director of his Company, he contributed greatly to the war effort by donating his services an appreciable part of each day to the training of Army and Navy doctors.

For more than a year he had devoted his after office hours far into the night making a most painstaking research of all material available on the subject of electrocardiographic findings. A few days prior to his death he completed "A Review of Electrocardiographic Interpretation". This compact, complete and excellent treatise was published by Northwestern University Medical School since his death, and it has received wide acclaim. Dr. Irwin possessed a fine analytical mind, a most unusual memory and a thoroughness rarely matched. He was a professional gentleman, whose life represented the very best in medicine, especially in the medicine of life insurance.

DR. WILLIAM M. JONES 1881-1944

It is with deep regret that we record the death of Dr. Wm. M. Jones, Medical Director of the Jefferson Standard Life Insurance Company, Greensboro, North Carolina, which occurred at his home on July 29, 1944, following an illness which began in the fall of 1943 and had incapacitated him since March.

Dr. Jones, a native of Wake County, North Carolina, was born January 23, 1881. He received his early education in Asheville, North Carolina, and his medical education at the University of Maryland, where he was graduated with the degree of M. D. in 1903. After serving an internship in Balti-

more, he entered private practice in 1905 in High Point, North Carolina. In 1912, he came to Greensboro, North Carolina as health officer of Guilford County in which position he distinguished himself in the field of public health. He came with the Jefferson Standard Life Insurance Company as Assistant Medical Director in 1923. In 1941, he was elected Medical Director, in which capacity he made an excellent record, serving until his death.

Public spirited and mindful of the welfare of others, Dr. Jones was active in civic affairs and projects for the betterment of his community. He was a faithful member of the Episcopal Church. As examining physician for the Local Draft Board in World War I and World War II, he freely contributed his time, skill and strength to his country. This he continued to do when, in the latter months, his strength was diminishing and time was growing short. This exemplifies the courage, faithfulness and unselfishness of the man.

His sterling qualities and good humor attracted to him a host of loyal friends. He was always a good companion. He enjoyed his friends and they enjoyed him. His business associates loved him for his kindness and friendliness and respected and admired him for his professional knowledge and good judgment. As a medical director, he was outstandingly successful. He is sorely missed by his friends and associates.

Dr. Jones is survived by his wife, the former Miss Lala Mundy, and three daughters, Mrs. Francis Jones Ernst, Miss Hortense Jones and Miss Patsy Jones, all of Greensboro, North Carolina.

DR. CARL LOVELACE

1876-1944

Dr. Carl Lovelace, of Waco, Texas, died on September 14, 1944, at his home, following an extended illness.

Dr. Lovelace was born February 6, 1876, in Crystal City, Missouri, the son of Thomas Jefferson and Sarah (Harrison)

Lovelace. The family moved to Waco in 1891, where he received his early education in the public schools and the Academy Department of Baylor University, from which he was graduated in 1894. He received an A.B. degree from Baylor University in 1898. Immediately after his graduation, he joined Roosevelt's Rough Riders of the United States Army, in San Antonio, and served with this famous organization in Cuba during the Spanish-American War. At the close of the war, he returned to Waco, and in 1899, attended the Medical Department of Vanderbilt University in Nashville, Tennessee, for one year. He later was graduated from George Washington University with an M. D. degree in 1902. He served his internship at George Washington University Hospital, at the completion of which he went with Surgeon General Gorgas to the Canal Zone, where he remained until the completion of the Panama Canal. In 1907, he completed a course in tropical medicine in the University of Liverpool, England, after which he became Chief Surgeon of the Madeira-Mamore Railroad in Brazil, for which position he was recommended by General Gorgas, because of his knowledge of tropical diseases. After construction of this railroad was finished, he accepted a position as surgeon with the Inca Mining Company in Peru. In 1913, Dr. Lovelace returned to Waco and entered the general practice of medicine.

In 1918, Dr. Lovelace was commissioned a Major in the Medical Reserve Corps of the United States Army, and during World War I, was stationed at Fort Oglethorpe, Georgia. At the close of the war he returned to private practice in Waco. In 1920, he was elected Medical Director of the Amicable Life Insurance Company, of Waco, Texas, which position he held until his death.

Dr. Lovelace had been a member of the McLennan County Medical Society, State Medical Association, and American Medical Association throughout his professional career in this state. He was past President of the McLennan County Medical Society. He was a Charter Member of the Texas Club of In-

ternists and had served that organization as President from February 1940 to February 1941. He was a member of the staff of the Central Texas Baptist Hospital and Providence Sanitarium. He was the author of a number of scientific articles, especially dealing with tropical diseases. In addition to his professional responsibilities, Dr. Lovelace found time to take a prominent part in the civic affairs of Waco. He served two years as a member of the City Commission of Waco, and at the time of his death, was Chairman of the Board of Directors of the Waco Public Library. He was a member of the Baptist Church and a Mason. Dr. Lovelace was held in the highest esteem by all who knew him. He was never too busy to assist any one who sought his help. His life was full and useful, and he carried on actively until his final illness.

Dr. Lovelace was married to Miss Lucille Hill of Waco, who died before him. He is survived by three sons.

DR. LEWIS FRANCIS MacKENZIE

1871-1944

Dr. Lewis Francis MacKenzie was born May 18, 1871 at Richmond, Quebec, the son of a Scotch father and an Irish mother, and throughout his life evidenced the charming characteristics of both those races. He attended St. Francis' College, and in 1894 was graduated in medicine from McGill University. For thirteen years he was actively engaged in the practice of medicine, came to the Home Office of the Prudential from Wellfleet, Massachusetts, in 1908, and served as Assistant Medical Director, and then Associate Medical Director until his retirement in 1941. He became a member of this Association in 1915 and a Member Emeritus in 1941. Following his retirement he lived in Burlington, Vermont, for two years, and, although an American citizen, spent the last year of his life in his native land, dying there on September 24, 1944. His widow, a sister, a daughter, and three grandchildren survive him.

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Dr. MacKenzie had a great interest in the subject of blood pressure and its relationship to insurance selection, having been actively engaged in study and research of that problem since about 1913. He contributed papers on this subject to the Association's Proceedings and to other publications.

Very deeply implanted within him were the doctrines of Presbyterianism and he lived his life with a strong realization of his Christian obligations. During a period of over thirty years, he served as an elder of the various Presbyterian churches of which he was a member and taught several Bible classes, where his gift of extemporaneous speaking and his extensive Biblical knowledge made his classes particularly interesting and delightful. He was an enthusiastic fisherman, and loved to tend his flower garden.

Those who had been associated with him over the years felt a great loss in his passing and will ever remember his kind and inspiring nature.

DR. RALPH BEVERLY OBER

1879-1945

Dr. Ralph Beverly Ober, Associate Medical Director of the Massachusetts Mutual Life Insurance Company, died on April 13, 1945, in Sarasota, Florida of coronary thrombosis.

Dr. Ober was graduated from the Harvard Medical School in 1901. In January, 1910, he was appointed Assistant Medical Director and in January, 1920, he was elected Associate Medical Director.

During his long association with us, his knowledge and experience were a distinct asset to our medical department even though, because of his widely recognized skill as a surgeon, the demands upon his time were too great to allow him to give his full services to our organization. But he proved a valuable aid as a consultant and advisor and gave freely of his time whenever possible.

Dr. Ober served as Major in the Medical Corps during the first World War and had an important assignment at one of the camps in the South.

He was a former president of the Springfield Hospital Staff and also took an active part in social welfare work in the city. He was especially prominent in the movement for the establishment of more playgrounds, being deeply interested in anti-tuberculosis work.

He was a member of the American Medical Association, Springfield Academy of Medicine, Clinical Club, New England Surgical Society, American Board of Surgery, and was a Fellow of the American College of Surgeons.

DR. JACOB ALLEN PATTON

1866-1944

Dr. Jacob Allen Patton, an emeritus member of this Association, died on September 25, 1944 at his home in Los Angeles following a very brief illness. The son of a physician, he was born in Charleston, Illinois, September 29, 1866. He graduated from the University of Illinois in 1888 and received his degree in medicine from Rush Medical College two years later. The following year was spent in his native town assisting his father with his practice, and he then returned to Chicago where he established himself in the practice of medicine and concurrently teaching chemistry at his alma mater. His thirty-eight years of active association with The Prudential Insurance Company began in 1895, when he became a medical examiner in Chicago. In 1908 he was invited to join the Home Office Medical Staff of the Prudential and filled successively the offices of Assistant Medical Director, Associate Medical Director, Medical Director and Second Vice President and Medical Director, retiring from active service in 1933.

He became a member of this Association in 1909 and was active in its affairs during his twenty-four years of membership, contributing a number of scientific papers and occupy-

ing positions on several important committees. He served as President for the term 1928-1929.

Dr. Patton, naturally of a friendly and sociable disposition, cultivated a wide acquaintance in life insurance circles, took a helpful interest in the problems of others and was well known and loved by many in this Association. This interest was maintained during the eleven years of his retirement, and his enthusiasm for the climate and life of California and his interest and love for old friends in the east caused him to cross the continent many times.

He is survived by his widow, Mrs. Kate W. Patton, and by a grandson. We all regret his passing and shall miss his friendship.

DR. WILLIAM O. PAULI

1882—1944

Dr. Pauli died suddenly at his home in Cincinnati, Saturday, September 16. Apparently in excellent health, he had just completed some work on his lawn when he was stricken with a heart attack from which he could not be revived.

Dr. Pauli was born in Cincinnati, February 11, 1882. He completed his elementary schooling in this city, and then entered the University of Cincinnati from which he graduated in 1902 with a Bachelor of Science degree. He taught biology for a year, and then entered Johns Hopkins University, getting his M.D. in 1907. After interning at Johns Hopkins Hospital, he took up the practice of medicine in Cincinnati.

He joined the Medical Staff of the Union Central in 1910 and in 1911 was made Assistant Medical Director. He held this post until February, 1942, when he was elected Associate Medical Director. During his long period of service with the Union Central, he was instrumental in establishing the Company's chemical laboratory, in compiling many valuable statistical studies in the field of underwriting and also in setting up a smooth-working procedure for the selection of Medical Examiners.

A faithful member of the Seventh Presbyterian Church of Cincinnati, Dr. Pauli also belonged to the Lion's Club, Cincinnati Academy of Medicine, Ohio State Medical Association, American Medical Association, American Public Health Association, and the Heart Council of the Public Health Federation of Cincinnati. He was a member for many years of the Association of Life Insurance Medical Directors.

He is survived by his wife, Mrs Winifred Weeks Pauli, and two daughters, Mrs. William J. Maynard and Miss Virginia Pauli.

Dr. Pauli had a host of friends, both in and out of his profession. Always a student, he was remarkably well informed in all branches of medicine. He was kind and generous and loyal, and his passing will be mourned far into the future by his associates and admirers.

DR. JAMES A. ROBERTS

1876—1945

Dr. James A. Roberts (born August 18, 1876), died suddenly of a heart attack on July 23, 1945.

He was a graduate in medicine, University of Toronto, and a Fellow of the Royal College of Surgeons of England. Shortly after qualifying for his fellowship in surgery, he joined the Canadian army and served in the South African War in 1900 and 1901. On his return he was appointed to the surgical staff of the Toronto General Hospital.

When the first World War broke out in August 1914 he was given command of the University of Toronto Hospital, known as No. 4 Canadian General Hospital, which he took to England and later to Salonica. Later he was appointed inspector of hospitals in England for the Canadian forces. For his military service he was appointed a Commander of the Bath.

On returning from overseas in 1919 he continued in practice until his appointment as Assistant Medical Director of The Canada Life Assurance Company in October 1925.

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His hobbies were golf and hunting. He greatly delighted in the northern woods of Ontario, which he visited every year in the hunting season.

He had a real capacity for friendship and was highly esteemed by those who knew him well. His loss is particularly mourned by those of us in this office who were his close associates.

DR. ROBERT SANDERSON

1904—1945

Dr. Robert Sanderson, Assistant Medical Director of the John Hancock Mutual Life Insurance Company, was born in Ayer, Massachusetts, on January 20, 1904.

He prepared for college at the Groton School, and was graduated from Yale in 1926. After graduation he taught for two years and then entered Harvard Medical School with the Class of 1932. On receiving his Medical Degree, he served at the Beverly Hospital for six months, and then took a regular eighteen months' service at the Boston City Hospital.

Dr. Sanderson was in general practice in Dedham, until he was appointed Assistant Medical Director on October 15, 1939. When the war broke out, he was commissioned a Lieutenant in the U. S. Naval Reserve, and was assigned to the Bureau of Medicine and Surgery at Washington, where his knowledge of medical selection could be utilized. He died suddenly on February 20, 1945, and was buried in the National Cemetery, Arlington, Virginia.

Although Dr. Sanderson was with the Company only three years, he showed excellent knowledge of insurance problems. He was a conscientious worker and always cheerful and friendly. He was devoted to his family and interested in community affairs and will be greatly missed by his many friends and business associates.

DR. ARTHUR L. SHERRILL

1871—1944

It is with deepest regret that the Equitable Life Assurance Society announces the death of Dr. Arthur L. Sherrill, Assistant Medical Director, at Chicago, on December 2, 1944. Born in Clinton, New York, in 1871, he received his degree of Doctor of Medicine, from New York University in 1898, and served as interne at Bellevue Hospital from 1898 to 1903.

Dr. Sherrill began his service with the Society in 1904, as a Medical Examiner at the Home Office. In 1911, he was appointed Assistant Medical Director, and in 1914, was transferred to Chicago, in charge of the Society's Medical office there.

His unfailing cheerfulness, conscientious devotion and loyalty, as well as his sterling character and lovable personality, greatly endeared him to his associates in the field and at the Home Office.

Those who had the privilege of knowing Dr. Sherrill all feel a deep and personal loss in his passing.

DR. CHARLES H. WILLITS

1857—1942

After nearly a half-century of continuous association with the Provident Mutual Life Insurance Company, Dr. Charles H. Willits left his desk in the Medical Department on September 16, 1940, and went with Mrs. Willits to a comfortable cottage in quiet and attractive surroundings in Coconut Grove, Florida, to live. It was there, after only a few days' illness, that he died on January 2, 1942.

Doctor Willits was born in Philadelphia on February 27, 1857. He was graduated from the University of Pennsylvania Medical School in 1879. For a number of years he was associated in practice with Dr. W. W. Keene, a Philadelphia surgeon of distinction. Later he established his own private practice in Philadelphia, and started to examine for the Provident in 1892.

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In 1902 he became a member of the Home Office staff—and, in 1906, was appointed medical director. Under his leadership the medical affairs of the Company were capably managed. For a while he was the only physician in the department. As the volume of business increased, it became necessary to add one doctor, then another—so that at the time of his retirement there were four doctors in the department.

In 1936 the Board of Directors gave Doctor Willits the title of medical adviser—and, for the next four years, he continued to come to the office almost daily. During those last years many questions of policy were discussed with him, and his interest in Company affairs was always very keen.

Throughout his long association with the Company he was beloved by officers, the field force, and employees alike. His position as medical director brought him into especially intimate relationship with the field organization, who regarded him with confidence and affection. His was a rare personality which attracted the casual acquaintance as well as his intimate friends. In his official capacity it was often necessary to take action which would cause disappointment to agent and client. He had ability which enabled him to decline an application with finality that gave no occasion for misunderstanding, yet with a sympathy and grace that would invariably win respect and good will. This quality of sympathetic understanding of every human frailty, as well as those qualities in men and women which make for success, won for him the confidence and respect of all who knew him. He was completely free from egotism—and, with a mildness of manner and an arresting twinkle in his eye, he could put a too-certain individual in his place, and with a few words bring into line refractory behavior. He had a clear insight which quickly distinguished first things first. He knew when to be rigid in decision and when to be generous—and he was always gracious.

In the fulness of a rich life Doctor Willits has passed from among us. Although we will not see him again—his memory, fragrant with countless kind deeds and words, will remain with us as the years go by.

DECEASED MEMBERS

John L. Adams, M. D.	New York, N. Y.
Charles D. Alton, M. D.	Hartford, Conn.
Malcolm O. Austin, M. D.	San Francisco, Calif.
Walter C. Bailey, M. D.	Boston, Mass.
Henry A. Baker, M. D.	Kansas City, Mo.
A. W. Barrows, M. D.	Hartford, Conn.
John T. J. Battle, M. D.	Greensboro, N. C.
Wesley W. Beckett, M. D.	Los Angeles, Calif.
Charles D. Bennett, M. D.	Newark, N. J.
Emmanuel P. Benoit, M. D.	Montreal, Can.
Charles Bernacki, M. D.	New York, N. Y.
Thomas W. Bickerton, M. D.	New York, N. Y.
Albert W. Billing, M. D.	New York, N. Y.
Wilton F. Blackford, M. D.	Louisville, Ky.
David N. Blakely, M. D.	Boston, Mass.
Robert J. Blanchard, M. D.	Winnipeg, Man.
Harold E. Bogart, M. D.	New York, N. Y.
Frederick G. Brathwaite, M. D.	New York, N. Y.
William R. Bross, M. D.	New York, N. Y.
Chauncey R. Burr, M. D.	New York, N. Y.
Robert L. Burrage, M. D.	Newark, N. J.
James Campbell, M. D.	Hartford, Conn.
Willard B. Carpenter, M. D.	Columbus, Ohio
Frank W. Chapin, M. D.	New York, N. Y.
Frederick W. Chapin, M. D.	Springfield, Mass.
Ferdinand E. Chatard, M. D.	Baltimore, Md.
Charles L. Christiernin, M. D.	New York, N. Y.
Henry Colt, M. D.	Pittsfield, Mass.
Henry W. Cook, M. D.	Minneapolis, Minn.
Thomas C. Craig, M. D.	New York, N. Y.
Hamilton C. Cruikshank, M. D.	Toronto, Canada
Edward Curtis, M. D.	New York, N. Y.
Clark W. Davis, M. D.	Cincinnati, Ohio
William B. Davis, M. D.	Cincinnati, Ohio
Charles A. Devendorf, M. D.	Detroit, Mich.

DECEASED MEMBERS

Henry K. Dillard, M. D.	Philadelphia, Pa.
Frank Donaldson, M. D.	Baltimore, Md.
Percy G. Drake, M. D.	Hartford, Conn.
Edwin W. Dwight, M. D.	Boston, Mass.
James B. Eagleson, M. D.	Seattle, Wash.
Z. Taylor Emery, M. D.	New York, N. Y.
Joseph E. Engelson, M. D.	New York, N. Y.
Francis C. Evers, M. D.	New York, N. Y.
William G. Exton, M. D.	Newark, N. J.
Robert H. Feldt, M. D.	Milwaukee, Wis.
John W. Fisher, M. D.	Milwaukee, Wis.
Paul FitzGerald, M. D.	Newark, N. J.
Thomas A. Foster, M. D.	Portland, Me.
Robert A. Fraser, M. D.	New York, N. Y.
Samuel W. Gadd, M. D.	Philadelphia, Pa.
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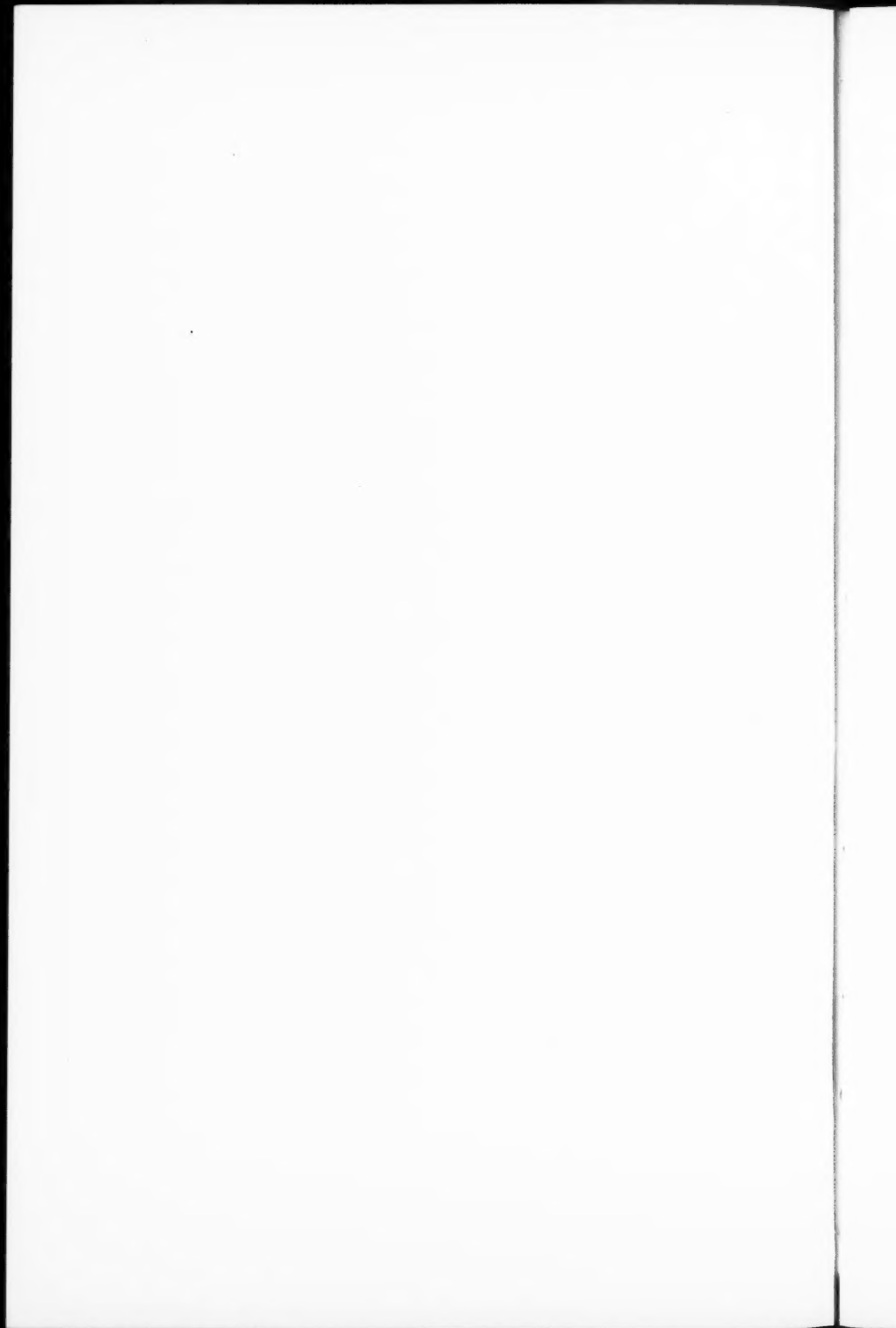
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